



**PHD**

**Effect of Acute Exercise on Energy Intake, Physical Activity Energy Expenditure and Energy Balance Hormones in Sedentary and Active Men**

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# **EFFECT OF ACUTE EXERCISE ON ENERGY INTAKE, PHYSICAL ACTIVITY ENERGY EXPENDITURE AND ENERGY BALANCE HORMONES IN SEDENTARY AND ACTIVE MEN**

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A thesis submitted for the degree of Doctor of Philosophy

University of Bath

School for Health

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## ABSTRACT

An exercise-induced energy deficit may affect post-exercise energy intake, physical activity energy expenditure (PAEE) and energy balance hormones. Therefore, the objective of this thesis was to investigate the impact of a single bout of exercise either of moderate (40%  $\dot{V}O_2\text{max}$ ) or high (70%  $\dot{V}O_2\text{max}$ ) intensity on post-exercise energy intake, physical activity energy expenditure and energy balance hormones in both sedentary and active males.

Physical activity energy expenditure increased between 38 and 62 hours following moderate intensity exercise in sedentary males (Chapters 3 and 4). This was due to increased light intensity energy expenditure (2.4-4.79 METs) such as standing and walking activities (Chapter 4). The Change in PAEE was not associated with circulating leptin and adiponectin concentrations. There was no impact of a single bout of exercise on post-exercise energy intake in sedentary males during a buffet meal (Chapter 4).

Chapter 5 aimed to determine whether changes in PAEE, energy intake, and energy balance hormones were related to physical activity status. Interestingly, plasma acylated ghrelin concentration was suppressed while total peptide YY (PYY) concentration tended to be elevated after high intensity exercise in active males (Chapter 5). However, there was no impact of either moderate or high intensity exercise on PAEE and post-exercise energy intake in active males.

The final study (Chapter 6) determined whether high intensity exercise in the fed state after a few days of food restriction had an impact on circulating energy balance hormones. Circulating postprandial total PYY and pancreatic polypeptide (PP) were increased for one hour after high intensity exercise in active men. There was no change in PAEE and post-exercise energy intake after exercise.

## **PUBLICATIONS AND CONFERENCE PROCEEDINGS**

### **CONFERENCE PROCEEDINGS**

Silalertdetkul, S., Stokes, K., Thompson, D. (2008). A single bout of moderate intensity exercise increases free-living physical activity energy expenditure over the following three days in sedentary males. Accepted for communication at the 13<sup>th</sup> ECSS Congress, Estoril.

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## ABBREVIATIONS

AEE	Activity energy expenditure
ANOVA	Analysis of variance
AUC	Area under the curve
BMC	Bone mineral content
BMI	Body mass index
BMR	Basal metabolic rate
BP	Blood pressure
CCK	Cholecystokinin
DLW	Doubly labelled water
EDTA	Ethylenediaminetetraacetic acid
EE	Energy expenditure
EI	Energy intake
ELISA	Enzyme-linked immunosorbent assay
FFA	Free fatty acid
GLP-1	Glucagon like peptide 1
Hb	Haemoglobin
HR	Heart Rate
HR <sub>max</sub>	Maximum heart rate
kcal	Kilocalorie
Kg	Kilograms
METs	Metabolic equivalents
ml	milliliter
NEAT	Non-exercise activity thermogenesis
ng	nanogram
nmol	nanomol
$\dot{V}O_2$	Oxygen consumption
$\dot{V}O_{2max}$	Maximum oxygen consumption
PAEE	Physical activity energy expenditure
PAL	Physical activity level
PAR-Q	Physical activity readiness questionnaire
pg	picogram

PP	Pancreatic polypeptide
PYY	Peptide YY
REI	Relative energy intake
RER	Respiratory exchange ratio
RM	Repetition maximum
RMR	Resting metabolic rate
RPE	Rating of perceived exertion
SD	Standard deviation
SE	Standard error
SPSS	Statistical package for the social sciences
SV	Stroke volume
TAG	Triglyceride
TDEE	Total daily energy expenditure
TEE	Total energy expenditure
TEF	Thermic effect of food
VAS	Visual analogue scales
W	Watt

## **CHAPTER 1**

### **INTRODUCTION AND LITERATURE REVIEW**

#### **1.1 Introduction**

Obesity and being overweight is a major health problem in both developed and developing countries. Increased adiposity is strongly implicated in many diseases, such as hypertension, diabetes mellitus, stroke, ischemic heart disease, breast cancer and osteoarthritis (Haslam and James 2005). Obesity and being overweight is the consequence of an energy imbalance between energy expenditure and energy intake. Increases in energy intake and insufficient physical activity or sedentary behaviour are the cause of obesity. An effective way to lose weight is to decrease daily energy intake and increase daily energy expenditure through physical activity (or both). However, exercise intervention are only modestly successfully of losing weight (Shaw et al. 2006). Furthermore, it is difficult to maintain a weight reduction programme in the long term with only around ten percent being successful (Unick et al. 2009). Clearly, research into the effects of exercise on feeding behaviour and energy balance are very important in order to understand the role of exercise in the prevention of obesity and obesity-related diseases.

The most malleable component of energy output or energy expenditure is physical activity or exercise. Exercise can increase energy expenditure directly and might also influence energy intake and physical activity energy expenditure indirectly through changing the secretion and concentration of hormones that are involved in the regulation of energy intake and physical activity energy expenditure (McMurray and Hackney 2005). There is emerging evidence that an acute bout of exercise can modify the concentration of certain 'energy balance hormones' (McMurray and Hackney 2005) and further work is required to build on these early observations. Although speculative at the present time, it is possible that the regulation of post-exercise energy intake in active individuals might be better than in sedentary individuals and this might help maintain an appropriate body weight. However, little is known about whether active men respond to a single bout of exercise differently to sedentary men. Furthermore, it is still unclear

whether the effect of acute exercise is influenced by energy restriction (dieting) or meals prior exercise. There are numerous other related research questions, for example, whether post-exercise energy intake, physical activity energy expenditure and energy balance hormones are influenced by exercise intensity. Therefore, the broad aim of the work described in this thesis was to investigate the interaction between a single bout of exercise and post-exercise energy intake, physical activity energy expenditure and energy balance hormones. This review focuses on several areas that provide the context for this work; such as regulation and measurement of energy intake and energy expenditure and what has already been established regarding the impact of acute exercise on energy balance (energy intake, energy expenditure and energy balance hormones).

## 1.2 Energy balance

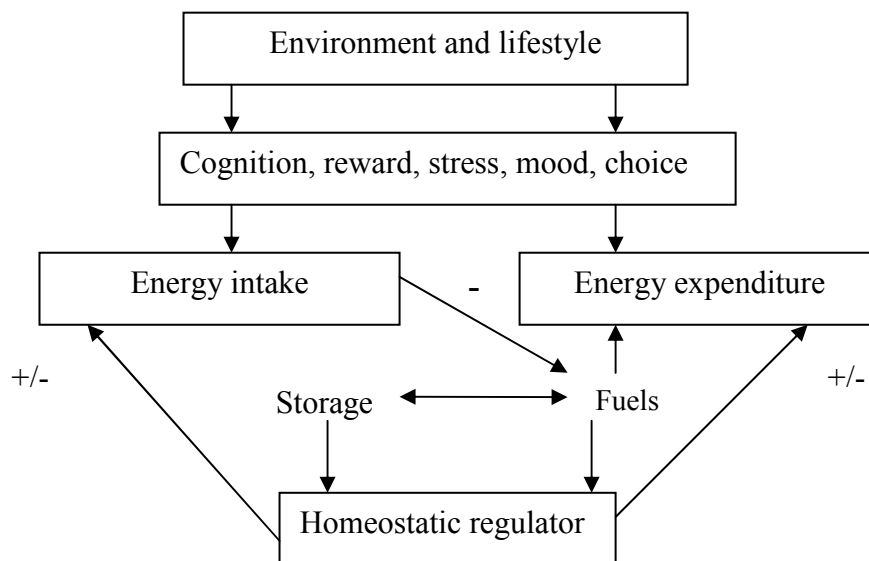


Figure 1.1 Factors regulating energy balance (Lenard and Berthoud 2008).

Energy can neither be created nor destroyed but may be converted from one form to another according to the first law of thermodynamics (Konturek et al. 2005). Energy balance is the product of energy intake and energy expenditure (Lenard and Berthoud 2008). For energy balance, energy intake must be equal to energy expenditure. For negative energy balance, energy intake must be lower than energy expenditure and for positive energy balance, energy intake must be greater than energy expenditure (Konturek et al. 2005). This relationship is complex (Figure 1.1) since there are

numerous factors that influence energy balance including environment, life style and genetics (Lenard and Berthoud 2008).

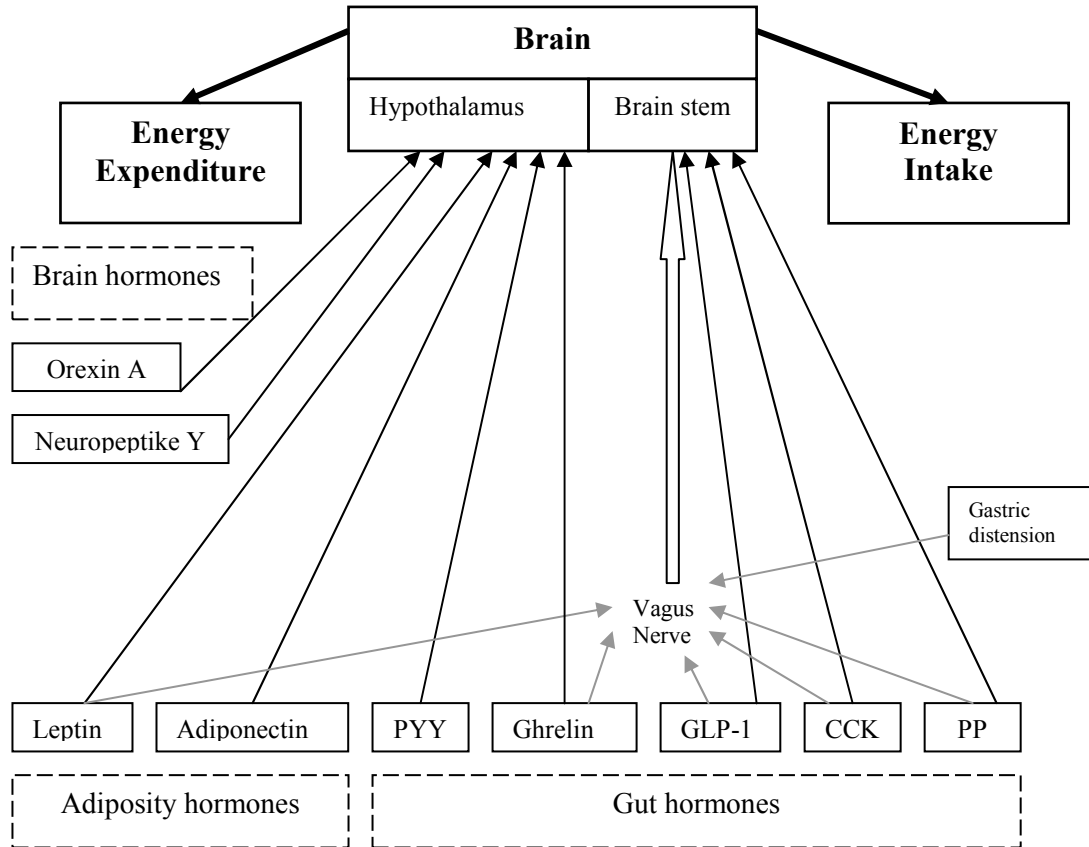


Figure 1.2 Model of central and peripheral hormones regulating energy balance. PYY, peptide YY; GLP-1, glucagon-like peptide 1; CCK, cholecystokinin; PP, peptide YY. This model is modified from Stanley et al. (2005) and Konturek et al. (2005).

Energy intake and energy expenditure are controlled by several hormones which are released not only from the brain but also from other areas such as adipose tissue and gastrointestinal tract (Figure 1.2; Stanley et al. 2005; Konturek et al. 2005). Orexin A and neuropeptide Y increase energy intake and energy expenditure (Stanley et al. 2005; Konturek et al. 2005). Ghrelin is released from the gut and increases energy intake and decreases energy expenditure (Stanley et al. 2005; Konturek et al. 2005). Leptin is released from adipocytes and increases energy expenditure and also inhibits energy intake (Stanley et al. 2005; Konturek et al. 2005). Total PYY, PP and GLP-1 are secreted from the gastrointestinal tract and inhibit energy intake (Stanley et al. 2005; Konturek et al. 2005).

### 1.3 Energy intake

Energy intake or food intake is determined by appetite, which includes hunger, satiation and satiety (Mattes et al. 2005). Hunger is the sensation that encourages food consumption and is influenced by metabolic, sensory and cognitive aspects (Mattes et al. 2005). Satiation is the process that happens following eating in order to control meal size (Blundell and Macdiarmid 1997). Satiety results from food consumption in order to determine the quantity of food and control subsequent hunger and eating (Blundell and Macdiarmid 1997).

#### 1.3.1 Regulation of energy intake

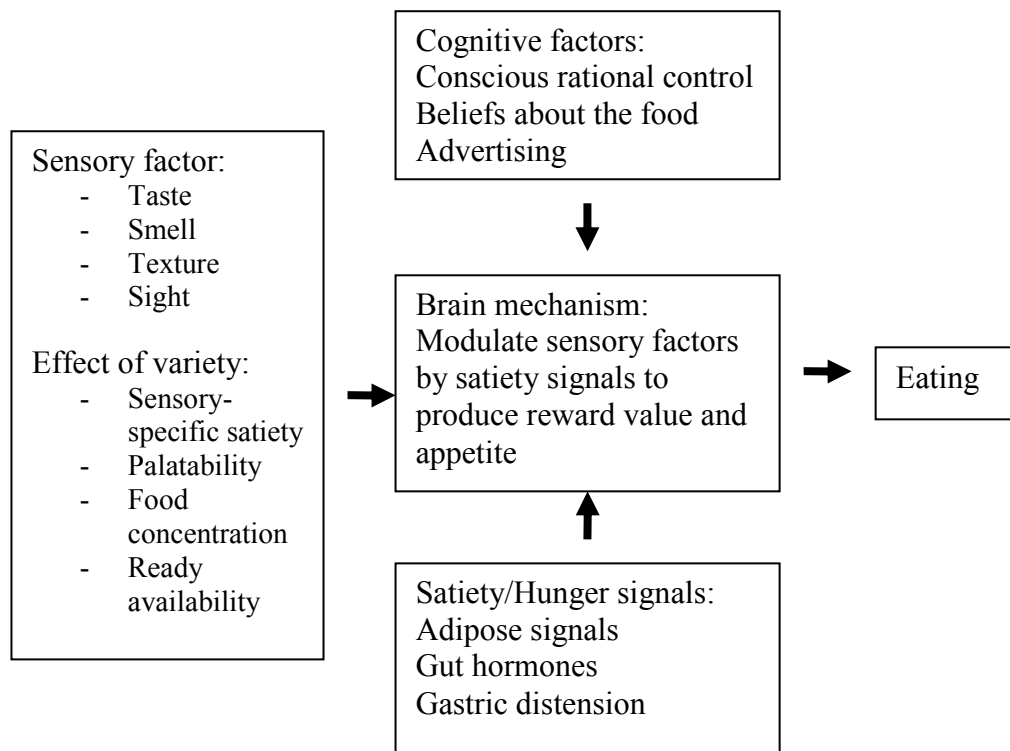


Figure 1.3 Interaction in the brain of sensory, cognitive and signal satiety and hunger interact to regulate energy intake (Rolls 2007).

Figure 1.3 shows sensory, cognitive and signal satiety and hunger interaction in the brain in order to determine eating behaviour (Rolls 2007). It appears that there are several factors that influence eating behaviour. For example, hormones from the hypothalamus and gut influence hunger and satiety. Peptide YY, pancreatic polypeptide, cholecystokinin, glucagon like peptide and serotonin are examples of satiety hormones

(Huda et al. 2006). While ghrelin, orexin A and B, and neuropeptide Y are the examples of hunger hormones (Huda et al. 2006).

There are two systems which influence energy intake (Konturek et al. 2005). Short-term regulation is associated with preventing overeating at each meal (Konturek et al. 2005). Regulation of energy intake at a single meal depends on volume, energy content and duration (Konturek et al. 2005). Following food ingestion, a signal from oropharyngeal and gastric areas are conveyed to neurotransmitters in the brain stem through afferent nerves stimulating satiety (Konturek et al. 2005). Moreover, mechanical distension and various hormones which are released from the gastrointestinal mucosa contribute to the peripheral signalling to the hypothalamus (Konturek et al. 2005). Long-term regulation is associated with the maintenance of normal quantities of energy stores such as body fat (Konturek et al. 2005). Ghrelin is an example of hormone released mainly from the stomach and which is involved in short-term regulation of energy intake (Konturek et al. 2005). Leptin and insulin are examples of hormones released from adipocytes and the pancreas which are involved in long-term regulation of energy intake (Konturek et al. 2005).

### **1.3.2 Measurement of energy intake**

Energy intake includes all food and beverage consumption (Rutishauser 2005). There are various methods available for the estimation of energy intake. For example, 24 hour recall is an inexpensive and easy method used to assess average normal food intake in large populations (Gibson 2005). However, this depends to a great degree on participant memory (Gibson 2005). Food frequency questionnaires assess the frequency of consumption of a given food item and food group and provides descriptive qualitative information about normal food consumption patterns (Gibson 2005). Estimated food intake consists of recording all food and beverage for seven days, with energy intake being calculated using food composition data. Assessment of normal intake and accuracy depends on the ability of participants to estimate quantities and amounts (Gibson 2005). Weighed food and fluid records are time-consuming for the participant but provide the best measure of energy intake (Gibson 2005). It is important that information such as the method of food preparation, description of food, brand



name and weight are recorded (Gibson 2005). However, one danger is that the normal eating pattern is disturbed in order to make it easy to weigh the food item or to impress investigators (Gibson 2005). In addition, there is day-to-day variation of food intake and this might not always reflect long-term habitual energy intake (Rutishauser 2005). Nevertheless, there is good evidence that relatively short-term weighed food and fluid records provide information on macronutrient intake (Rutishauser 2005). Some of these issues can be overcome through the use of laboratory-based dietary studies where precise information on the food that has been consumed can be obtained (Rutishauser 2005). However, one limitation to this approach is that other aspects of behaviour (e.g., physical activity) will be disturbed.

Energy intake measurement
<b>Field methods</b>
Records
<ul style="list-style-type: none"><li>- Menu record</li><li>- Estimated record</li><li>- Weighed record</li></ul>
Recalls
<ul style="list-style-type: none"><li>- 24 hour recall</li><li>- Diet history</li><li>- Food-frequency questionnaire</li></ul>
<b>Laboratory methods</b>
Laboratory-based dietary studies
Analysis of food consumed
<ul style="list-style-type: none"><li>- Collect duplicate of all food</li><li>- Collect sample of food</li><li>- Reconstruct a composition from the record of food</li></ul>
Markers of energy and nutrient intake
<ul style="list-style-type: none"><li>- Urinary nitrogen</li><li>- Doubly labelled water method</li><li>- Energy intake/Basal metabolic rate ratio</li></ul>

Table 1.1 Energy intake measurement in laboratory and field method (Rutishauser 2005; Hollis et al. 2005; Gibson 2005; Mattes et al. 2005).

Other tools for the estimation of energy intake have also been proposed. For example, it has been suggested that urinary nitrogen excretion provides some information on protein intake (Rutishauser 2005). The doubly labelled water (DLW) method estimates total energy expenditure from CO<sub>2</sub> production (Rutishauser 2005). If there is no change in body weight, energy intake is equivalent to energy expenditure (Rutishauser 2005). It is accurate and suitable for free living conditions but it is expensive (Rutishauser 2005).

As an adjunct to the assessment of energy intake, some investigators have sought to capture information on appetite (Mattes et al. 2005). Visual analogue scales are the common method for the assessment of subjective feelings of hunger and fullness (Mattes et al. 2005). Alternatively, some investigators have proposed that selected biomarkers might provide information on appetite including the concentration of hormones such as ghrelin, leptin, glucagon like peptide 1 and cholecystkinin (Mattes et al. 2005). These measures are influenced by several factors such as nutrient levels, biological confounders such as genetic background, and the presence of other environmental factors such as the interaction with other changes in the gut during absorption and metabolism (Gibson 2005).

#### 1.4 Energy expenditure

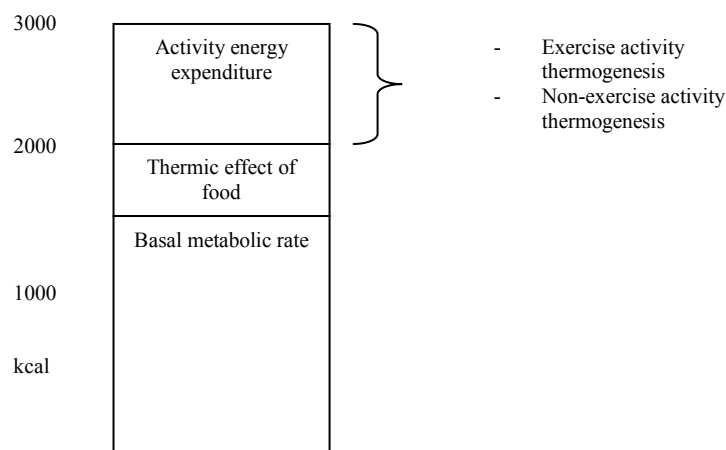


Figure 1.4 The components of total daily energy expenditure (Novak et al. 2007).

Total energy expenditure (TEE) is composed of three basic components: basal metabolic rate; thermic effect of food (TEF); and activity energy expenditure (Novak et

al. 2007). Figure 1.4 shows the components of total energy expenditure. Basal metabolic rate is the energy required for maintain body functions which is affected by many factors such as climate, genetics, body surface area, sleep, gender, body temperature, and the circulation of some hormones like leptin, thyroid and adrenaline concentrations (Ganong 1995). The thermic effect of food is the energy expended in the digestion, absorption and storage of energy. Activity energy expenditure (AEE) consists of exercise activity thermogenesis and non-exercise activity thermogenesis. Non-exercise activity thermogenesis (NEAT) is the energy expenditure in all activity not including sleeping, eating and exercise; and includes activities such as walking to work, shopping and typing (Levine and Kotz 2005). NEAT is impacted on by biological factors such as genes and energy balance hormones but also non-biological factors such as cognitive input, cultural and environmental issues includes occupation, travel to work and leisure time (Levine and Kotz 2005).

#### **1.4.1 Regulation of energy expenditure**

Energy expenditure processes are controlled by the brain (Dishman et al. 2006). Energy expenditure is composed of basal metabolic rate, dietary induced thermogenesis, spontaneous physical activity as well as exercise (Lenard and Berthoud 2008). Energy expenditure is influenced by either subconscious behaviour or conscious voluntary behaviour (Lenard and Berthoud 2008). Conscious voluntary behaviour, for instance voluntary exercise, is regulated by the motor cortex of the brain (Lenard and Berthoud 2008). Subconscious behaviour is both autonomic and regulated by endocrine stimuli (Lenard and Berthoud 2008). In humans, this is particularly complex because this is influenced by multiple other considerations such as environment, physical development and social factors (Dishman et al. 2006).

Circulating hormones such as leptin, thyroid hormones and adrenaline all influence basal metabolic rate (Ganong 1999). Adrenaline influences stimulation of glycogen and triglycerol catabolism and metabolic rate, for example with emotional stress (Ganong 1999). Thyroid hormones influence basal metabolic rate by increasing oxygen consumption and heat production of most body tissues (Ganong 1999). It also increases the production of uncoupling proteins in mitochondrial membranes (Ganong 1999).

Basal metabolic rate and appetite decrease when there is thyroid hormone deficiency whereas, conversely, basal metabolic rate, appetite and catabolism of nutrients increase when there is thyroid hormone excess (Ganong 1999).

The mechanisms underlying the impact of physical activity and exercise on the function of central and peripheral neural systems are still unclear (Dishman et al. 2006). It has been suggested that physical activity and exercise might influence the central nervous system through metabolic and neuro-chemical pathways between skeletal muscle, the spinal cord and brain (Dishman et al. 2006). Temporary increases in local cerebral glucose use in the motor cortex, cerebellum, thalamus and hypothalamus have been demonstrated following acute exercise in animals (Dishman et al. 2006). In humans, hypothalamus and motor cortex blood flow are increased during exercise (Dishman et al. 2006). Acute exercise might alter brain function and therefore induce change in certain behaviours.

As discussed earlier, non-exercise activity thermogenesis or NEAT is one of the major components of activity energy expenditure. There are many hormones that can influence NEAT. Orexin A (hypocretin 1) is one hormone released from the hypothalamus. Spontaneous physical activity increases when orexin A is injected into the paraventricular nucleus of rats (Kiwaki et al. 2003). It is possible that orexin A might influence NEAT in humans. Leptin is secreted mainly from adipocytes and also other areas such as stomach, liver, placenta, heart and skeletal muscle (Sandoval and Davis 2003; Bouassida et al. 2006). It acts on the hypothalamus (Sandoval and Davis 2003; Bouassida et al. 2006) and intracerebroventricular leptin administration increases spontaneous physical activity in rats (Choi et al. 2008). An inverse relationship between fasting leptin concentration and physical activity estimated using heart rate in humans has been reported (Franks et al. 2003).

There are also several other factors which influence NEAT. Thyroid hormone excess is associated with increased NEAT in animals (Levine et al. 2003). Estrogens increase spontaneous physical activity in mice while estrogen deficiency decreases spontaneous physical activity in mice and women (Kotz et al. 2008). Dopamine is a neurotransmitter associated with motor activity such as spontaneous physical activity (Kotz et al. 2008).

There are some hormones which have the capacity to decrease spontaneous physical activity in animals such as ghrelin and neuropeptide Y (Kotz et al. 2008).

#### **1.4.2 Measurement of energy expenditure**

Physical activity level (PAL) is the ratio between total energy expenditure and resting energy expenditure over 24 hours (Schutz et al. 2001). A high PAL is therefore the product of greater activity energy expenditure (AEE). Importantly, this represents the total cost of activity above rest and does not provide information on intensity and duration of activity (Schutz et al. 2001). There are numerous ways to express the intensity of exercise and physical activity (e.g. absolute oxygen cost or oxygen cost relative to maximum oxygen uptake). In physical activity contexts, Metabolic Equivalents or METs is a common method. One MET is equal to 3.5 ml of oxygen per kg of body mass and multiples of METs gives information on the intensity of a specific physical activity relative to body mass (Schutz et al. 2001).

There are several methods for monitoring activity energy expenditure in free-living conditions (Table 1.2). These include questionnaires, integrated multi-sensor monitors, accelerometers, pedometers, heart rate monitors, physical activity monitors and doubly labelled water (Casaburi et al. 2007; Bouchard et al. 2000). Physical activity questionnaires provide some information on a given individual but they are generally unreliable and each questionnaire will give a very different profile (Shephard 2003). Pedometers assess vertical oscillation and provide a measure of total movement but they are limited because they are unable to measure some movements such as cycling and slow walking speed, and also they provide no information on activity intensity (Bouchard et al. 2000). Accelerometers provide an objective indicator of total movement in terms of intensity and duration, however, accelerometers cannot detect some static activities such as lifting and cycling (Bouchard et al. 2000; Schutz et al. 2001). The advantages of these methods include that they are convenient, inexpensive and non-invasive (Schutz et al. 2001).

Heart rate monitors provide an objective indicator of the physiological effect of physical activity and energy expenditure is estimated from an equation of the relationship

between heart rate and oxygen consumption (Bouchard et al. 2000). There are some limitations of heart rate monitoring, which include day to day variation in heart rate. For example, heart rate may increase with no effect on oxygen consumption; perhaps as a result of high ambient temperature and humidity or emotion (Montoye et al. 1996). Fatigue and hydration state alter the relationship between  $\dot{V}O_2$  and HR. Arm activity causes a higher heart rate than leg activity with the same work load (Montoye et al. 1996). Additionally, the relationship between heart rate and oxygen consumption is relatively poor at low intensity activities (Bouchard et al. 2000).

Energy expenditure measurement
<b>Field methods</b>
Doubly labeled water
Heart rate monitor
Behavioural observation and time or motion analysis
Questionnaires and interview
Movement assessment devices
- Pedometers
- Accelerometers
- Integrated multi-sensor monitor
Combine movement and heart rate variability
- Actiheart ( Physical activity monitor)
<b>Laboratory methods</b>
Direct calorimeter
Indirect calorimeter
- Closed-circuit spirometry
- Open-circuit spirometry
Portable spirometry
Bag technique
Computerized instrumentation
Biomechanic method
- Photography
- Force platform

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Table 1.2 Methods for assessing energy expenditure (Casaburi et al. 2007; Bouchard et al. 2000; Brage et al. 2006; Montoye et al. 1996; McArdle et al. 2007).

Recently, a combined synchronised heart rate monitor with accelerometer has been developed that seeks to capitalise on the relative strengths of each measurement (Brage et al. 2006). This instrument uses a branch model equation to estimate energy expenditure above rest – weighted towards accelerometer or heart rate depending on a number of specific factors. This combined approach has been found to provide a more precise assessment of physical activity energy expenditure than either method used in isolation (Brage et al. 2006). The validity of this instrument has been investigated in a range of different activities – including some where other tools would often fail (Brage et al. 2006; Thompson et al. 2006).

## **1.5 Exercise and energy balance**

Exercise increases energy expenditure and may, therefore, impact upon energy intake. There are several factors that influence energy intake in response to exercise including intensity, mode and duration (King 1998).

### **1.5.1 Effect of acute exercise on energy balance**

Acute exercise alters body temperature, blood flow to in the brain, blood flow to the stomach, blood triglyceride concentration and several energy balance hormones. As a consequence of this, exercise might alter energy intake and appetite.

#### **1.5.1.1 Effect of acute exercise on energy intake and appetite**

There are several studies that have investigated the impact of acute exercise on post-exercise energy intake (Tables 1.3 and 1.4). For example, there was no difference in energy intake after exercise during a buffet meal between moderate and high intensity exercise when the energy that was expended during exercise was not different (Pomerleau et al. 2004; Imbeault et al. 1997). However, one study found that energy intake during a buffet meal after high intensity exercise was considerably less than after moderate intensity exercise when there was no attempt to control the amount of energy expended during exercise (Kissieff et al. 1990). These results perhaps indicate that the total energy expenditure during exercise might be important.

One study reported that post-exercise energy intake after high intensity exercise was greater than in a resting condition (Pomerleau et al. 2004) whereas another study (Kissileff et al. 1990) demonstrated that energy intake after high intensity exercise was far less than resting. Other studies have found that there was no change in energy intake after high intensity exercise when compared with resting (Erdmann et al. 2007; King et al. 1994; King and Blundell 1995). It seems that the impact of high intensity exercise on post-exercise energy intake remains unclear. To complicate matters further, there have been some reports that even when there was no difference in energy intake after high intensity exercise in a buffet meal there was a delayed increase in eating following high intensity exercise (King et al. 1994; King and Blundell 1995).

The results of moderate intensity exercise studies are also rather mixed. Some studies showed the impact of moderate exercise intensity post-exercise energy intake during a buffet meal. For instance, energy intake after moderate intensity exercise was considerably less than resting in a fasting state (George and Morganstein 2003). One study reported that there was no difference in post-exercise energy intake between moderate intensity exercise and resting in a fasting state (Pomerleau et al. 2004). Furthermore, moderate intensity exercise in a fed state has resulted in both increased (Martin et al. 2007) and decreased (Ueda et al. 2009a; Ueda et al. 2009b) post-exercise energy intake. The interaction between feeding status (fed/fasted) and the responses to exercise may play a role in subsequent energy intake and this warrants further investigation.

Rather than assessing energy intake, some studies have concentrated on ratings of hunger. Hunger scores were suppressed following high intensity exercise (70%  $\dot{V}O_2\text{max}$ ) for at least 30 minutes (Broom et al. 2007; Broom et al. 2009; Burns et al. 2007; King et al. 1994; King and Blundell 1995). Some studies have demonstrated the suppression of hunger without altered energy intake after high intensity exercise (King et al. 1994; King and Blundell 1995). Other studies have found no change in post-exercise energy intake with increased hunger scores. It appears that subjective feelings of hunger are suppressed by high intensity exercise but the relationship between subjective feelings of hunger and energy intake following exercise is still unclear.



Relative energy intake is the energy intake after accounting for energy expenditure during exercise. Relative energy intake after moderate and high intensity exercise considerably lower than resting (Pomerleau et al. 2004; Imbeault et al. 1997; King et al. 1994). Some studies have shown that relative energy intake after high intensity exercise was fractionally less than after low and moderate intensity exercise even when there was no difference in the amount of energy expended during exercise (Imbeault et al. 1997; King et al. 1994).

Only a relatively small number of studies have examined the impact of repeated bouts of exercise on energy intake. There was no change in energy intake and subjective feelings of hunger and fullness during two bouts of exercise over two days in physically active men (King et al. 1997). However, post exercise energy intake increased after daily running at 68%  $\dot{V}O_2\text{max}$  for five days in men and women (Staten 1991).

Several studies have investigated the impact of acute exercise on energy intake and appetite but the results still unclear (Tables 1.3 and 1.4). Most of the studies found an impact of high intensity exercise on subjective feelings of hunger but did not provide food following exercise. Therefore, it is difficult to interpret data to determine whether there was an impact of exercise on post-exercise energy intake or on subjective feelings of hunger alone. Some studies found high intensity exercise leads to a decrease in post-exercise energy intake. However, it is still unclear whether the impact on post-exercise energy intake is caused by high intensity exercise or because energy expenditure during high intensity exercise was greater than during moderate intensity exercise.

Subjects	Experiment design	Results	Reference
Healthy females (22 yrs)	Waking and running at 40% $\dot{V}O_{2peak}$ (65 mins) and 70% $\dot{V}O_{2peak}$ (37 mins) (same energy cost). A buffet meal at lunch time.	EI at lunch time, HIE > Resting REI HIE and LIE < Resting No change VAS	Pomerleau et al. 2004
Healthy males and females (24 yrs)	Cycling at LIE (50W) and HIE (100 W) for 30 mins. Sandwiches at lunch time.	No change EI No change VAS	Erdmann et al. 2007
Healthy males (23 yrs)	Cycling at 75% and 50% $\dot{V}O_{2max}$ for 30 mins after consumed breakfast. Test meal.	↓EI EI not different MIE-HIE	Ueda et al. 2009b
Moderately active males (24 yrs)	Waking and running at 75% $\dot{V}O_{2max}$ (34 mins) and 35% $\dot{V}O_{2max}$ (72 mins) (same energy cost). A buffet meal.	No change EI, no different LIE-HIE REI HIE < LIE and Resting No change VAS	Imbeault et al. 1997
Well trained males (21 yrs)	Running at 70% $\dot{V}O_{2max}$ for 60 mins, resistance exercise 80% RM for 90 mins.	↓Hunger after running, resistance	Broom et al. 2009
Healthy males and females (26 yrs)	Intermittent cycling at 65% maximum HR for 60 mins after consumed chocolate drink. A buffet meal.	↑EI, ↓REI ↓Hunger during exercise	Martins et al. 2007
Well trained male and females (25yrs)	Running at 73% $\dot{V}O_{2max}$ for 60 mins.	↓Hunger	Burns et al. 2007
Well trained males (21 yrs)	Running at 72% $\dot{V}O_{2max}$ for 60 mins	↓AUC hunger	Broom et al. 2007

Table 1.3 A summary of studies investigating the effect of an acute bout of exercise and post-exercise energy intake in healthy participants. EI, energy intake; REI, relative energy intake; VAS, subjective feeling of hunger and fullness; LIE, low intensity exercise; MIE, moderate intensity exercise; HIE, high intensity exercise; AUC, area under the curve. Age (means).

Subjects	Experiment design	Results	Reference
Healthy and lean males	Cycling at 70% $\dot{V}O_2$ max (30 mins) and 30% $\dot{V}O_2$ max (60 mins). Free-selection test meal after exercise.	No change EI lean, healthy REI HIE<LIE lean, healthy ↓VAS HIE lean, healthy	King et al. 1994
Obese and healthy males (22 yrs)	Cycling at 50% $\dot{V}O_2$ max for 60 minutes after consumed breakfast. Test meal.	↓EI, ↓REI obese, healthy EI, REI obese < healthy No change VAS	Ueda et al. 2009a
Obese and non obese males (25 yrs)	Cycling at 60%Wmax for two hours. A buffet meal.	↓EI, ↓hunger obese, non-obese EI not different between group	Westerterp-Plantenga et al. 1997
Non obese and obese females (22 yrs)	Cycling at 90 W and 30W for 40 minutes. Test meal.	EI HIE < MIE non-obese No change EI obese	Kissileff et al. 1990
Healthy and Lean males	Cycling and running at 70% $\dot{V}O_2$ max. Free selection of food (high fat and low carbohydrate or low fat and high carbohydrate).	No change EI lean, healthy ↓Hunger after cycling and running No change VAS cycling- running.	King and Blundell 1995

Table 1.4 A summary of studies investigating the effect of an acute bout of exercise and energy intake in lean versus obese participants. EI, energy intake; REI, relative energy intake; VAS, subjective feeling of hunger and fullness; LIE, low intensity exercise; MIE, moderate intensity exercise; HIE, high intensity exercise; AUC, area under the curve. Age (means).

### **1.5.1.2 Effect of acute exercise on energy expenditure**

There have been reported of no change in daily total energy expenditure due to a decrease in non-exercise physical activity energy expenditure after 8 weeks (Meijer et al. 1999) and 12 weeks (Goran and Perlman 1992) of training in older adults. However, Hunter et al. (2000) showed that daily total energy expenditure and physical activity energy expenditure increased after 26 weeks training in older adults. Additionally, resistance training caused an increased in daily energy expenditure due to an increase in resting metabolic rate and also physical activity energy expenditure (Hunter et al. 2000). The increase in total energy expenditure may be due to an increase in resting metabolic rate, PAEE and sympathetic nervous system activity (Poehlman et al. 2002).

A few studies have examined the impact of an acute bout of exercise on energy expenditure in sedentary and active participants. Energy expenditure estimated using a heart rate monitor between 1 and 7 p.m. after cycling at a moderate intensity exercise was higher than after high intensity exercise; but was not different from resting in obese boys (Kriemler et al. 1999). However, the energy expended during high intensity exercise was higher than during moderate intensity exercise. In addition, assessments of energy expenditure were only taken over eight hours. In a different study, it was demonstrated that daily PAEE estimated using an accelerometer (actigraph) did not change during four daily bouts exercise at 50-65%  $\dot{V}O_2$  peak either with or without energy replacement in sedentary and overweight males and females (Hagobian et al. 2008). There was no difference in heart rate using a heart rate monitor and physical activity using physical activity records on the day after exercise in men who did regular sport between 3-5 times per week (King et al. 1997). Stubbs et al. (2002) reported that there was no change in non-exercise energy expenditure estimated using a heart rate monitor on the same day as either medium exercise (two 40 minutes exercise session per day) or high exercise level (three 40 minutes sessions per day) in sedentary women (Stubbs et al. 2002). Additionally, non-exercise physical activity using heart rate monitoring and self report for eight days during four sessions of exercise was not altered in young males and females who were moderately active but not involved in regular exercise (McLaughlin et al. 2006). In general, these studies tend to suggest that a single bout of exercise has no impact on subsequent physical activity. However, the

techniques that have been used are limited and also the period of observation has usually been restricted to only the same day as the acute bout of exercise.

Resting metabolic rate is a key component of total energy expenditure. Aerobic and resistance exercise have been shown to induce an increase in resting metabolic rate on the day after performance of exercise (Jamurtas et al. 2004; Williamson and Kirwan 1997). This increase in resting energy expenditure may be explained by many factors, for instance, the change in circulating thyroid hormone concentration particularly triiodothyronine (T<sub>3</sub>), increased in sympathetic nervous system activity and increased in protein degradation and reparation following exercise (Jamurtas et al. 2004).

#### **1.5.1.3 Interaction between acute exercise and energy hormone**

Several studies have examined the impact of a single bout of exercise on energy balance hormones such as ghrelin, leptin, total PYY, PP and GLP-1 (Tables 1.5 and 1.6). Ghrelin is composed of 28 amino acid peptides (Murphy et al. 2006; Huda et al. 2006). It is synthesized predominantly in the cells of the stomach and acts on the hypothalamus (Figure 1.2, Small and Bloom 2004; Huda et al. 2006). Plasma ghrelin concentration is elevated two fold before a meal and falls back within one hour of eating (Cummings et al. 2001). Intravenous ghrelin infusion (5.0 pmol/kg/min) leads to increased energy intake of approximately 30% and increased subjective feelings of hunger in humans (Wren et al. 2001). Administration of ghrelin leads to increased food intake and decreased spontaneous physical activity in rats (Tang-Christensen et al. 2004). It has been suggested that ghrelin may be the first hormonal trigger which influences energy intake and suppresses non-essential energy expenditure such as spontaneous physical activity in order to conserve remaining energy (Castaneda et al. 2005). Ghrelin exists in two forms: nonacylated ghrelin and acylated ghrelin or active ghrelin (Small and Bloom 2004). Acylated ghrelin is able to cross the blood brain barrier and the circulating concentration decreases after food intake (Hosoda et al. 2004; Blom et al. 2006). Acylated ghrelin rapidly responds to both exercise and glucose ingestion (Broom et al. 2007; Broom et al. 2009; Burns et al. 2007; Hosoda et al. 2004). It has been reported that ghrelin concentration in physically inactive is higher than physically active boys and girls (Jurimae et al. 2009; Jurimae et al. 2007a). Fasting total ghrelin and acylated

ghrelin was lower in obese males than in normal weight adolescent males (Mackelvie et al. 2007). Total ghrelin decreases after food restriction and exercise in competitive body builders (Maestu et al. 2008). Total ghrelin increased after 12 week training in obese children (Kim 2008) and increased after one year of moderate intensity exercise in women (Foster-Schubert et al. 2005).

Several studies have investigated the impact of acute exercise on ghrelin concentration. Total ghrelin concentration is not altered by high intensity exercise in the fasting state (Burns et al. 2007; Marzullo et al. 2008). Additionally, there is no impact of exercise on postprandial total ghrelin concentration after moderate intensity exercise in fed state (Martin et al. 2007; Ueda et al. 2009a). However, some studies have shown that total ghrelin concentration is decreased after circuit training exercise in a fasting state (Ghanbari-Niaki 2006) but increased after maximum exercise in elite male rowers (Jurimae et al. 2007b). Hagobian et al. (2009) reported that total ghrelin concentration increased in fed state after four exercise sessions either with or without energy replacement in sedentary and overweight males and females. It has been suggested that changes in total ghrelin concentration might be associated with the extent of energy expended during acute exercise, as well as being influenced by the population and also the exercise protocol (Jurimae et al. 2007b). Interestingly, it has been demonstrated in previous studies that fasting acylated ghrelin concentration was suppressed by high intensity running and resistance exercise but that the effect was short-lived (Broom et al. 2007; Broom et al. 2009). Acylated ghrelin concentration was suppressed after vigorous intensity exercise in a fasting state in obese more than lean individuals (Marzullo et al. 2008). In contrast, there was no impact of either high intensity exercise or resistance exercise on postprandial acylated ghrelin concentrations (Broom et al. 2009). Additionally, it has been demonstrated that acylated ghrelin was increased after four bouts of daily exercise without energy replacement in women not men (Hagobian et al. 2009) and after five days of aerobic exercise in normal children more than obese children (Mackelvie et al. 2007). In general, these results appear to suggest that high intensity exercise in a fasting state suppresses acylated ghrelin concentration but that there is no exercise-induced change in postprandial acylated ghrelin concentration in a fed state.

Peptide YY (PYY) is composed of 36 amino acid linear peptides (Huda et al. 2006). PYY is released mainly from ileum, colon and rectum (Wynne et al. 2005). PYY increases after a meal and remains elevated for up to six hours (Stanley et al. 2005). There are two forms including PYY<sub>1-36</sub> and PYY<sub>3-36</sub> (Wynne et al. 2005). PYY<sub>3-36</sub> has a high affinity with the Y2 receptor in the hypothalamus and can circulate across the blood brain barrier (Huda et al. 2006). Administration of PYY<sub>3-36</sub> results in decreased food intake during a buffet meal and decreased ghrelin concentration in lean and obese men and women (Batterham et al. 2003). Total PYY concentration is correlated with energy expenditure using indirect calorimetry (Doucet et al. 2008). PYY<sub>3-36</sub> concentration in obese individuals is considerably less than normal weight men and women (Batterham et al. 2003). Postprandial PYY<sub>3-36</sub> was lower in obese than in lean women (Zwirska-Korczala et al. 2007). Fasting total PYY concentration was elevated after 32 weeks of exercise training in overweight adolescents (Jones et al. 2009).

Only a few studies have examined the impact of a single bout of exercise on PYY. Fasting and postprandial total peptide YY (PYY) concentrations increased after moderate and high intensity exercise (Broom et al. 2009; Martin et al. 2007; Ueda et al. 2009b). The effect of moderate intensity exercise on postprandial total PYY seems to be small and disappears after exercise in a fed state (Martin et al. 2007; Ueda et al. 2009a; Ueda et al. 2009b). It has been reported that exercise for 30 minutes results in an increase in PYY<sub>3-36</sub> after both moderate and high intensity exercise in healthy men (Ueda et al. 2009b). Interestingly, Broom et al. (2009) reported that eating after high intensity exercise induced an elevation in postprandial total PYY concentration and this remained elevated for a few hours following exercise. One study reported either exercise a prior to a meal or exercise after a meal tended to increase PYY concentration more than a meal alone (Chang et al. 2009). Ueda et al. (2009a) showed a greater impact of exercise on total PYY concentration in healthy men than in obese men. In general, these results suggest that high intensity exercise either before or after a meal will increase PYY response to the meal.

Pancreatic polypeptide (PP) has 36 amino acid peptides, is secreted from islets of Langerhans, pancreas and distal gastrointestinal tract (Stanley et al. 2005). PP cannot cross the blood brain barrier (Huda et al. 2006). It acts in the hypothalamus and

brainstem areas via vagal system (Huda et al. 2006). Intra-third cerebroventricular administration of PP leads to increased locomotor activity in animals (Nakajima et al. 1994). Intravenous administration of PP (10 pmol/kg/min) results in decreased food intake and appetite in a buffet meal and dinner meal (food recorded) in healthy volunteers (Batterham et al. 2003). Fasting plasma PP concentration has been reported to be slightly elevated after ten weeks of exercise training at 70%  $\dot{V}O_2\text{max}$  (Hurley et al. 1991). It has been demonstrated that postprandial pancreatic polypeptide (PP) concentration increased after incremental and moderate intensity acute exercise (Martin et al. 2007; Sliwowski et al. 2001; Greenberg et al. 1986). It appears that exercise in a fed state increases PP to a greater extent than exercise in a fasted state (Sliwowski et al. 2001). Fasting and postprandial PP concentration was increased after three hours of cycling at 40%  $\dot{V}O_2\text{max}$  (Hilsted et al. 1980)

Leptin is the product of the ob gene, which has a molecular mass of 16 kDa (Bouassida 2006). It is secreted mainly from adipocytes and also from other areas such as the stomach, liver, placenta, heart and skeletal muscle (Sandoval and Davis 2003; Bouassida 2005). It acts on the hypothalamus and its main function is stimulating activity energy expenditure, suppressing food intake in the long term, increasing gluconeogenesis and glucose uptake and fat metabolism (Sandoval and Davis 2003; Bouassida 2005). Leptin also decreases fatty acid synthesis (Sandoval and Davis 2003). The concentration of leptin depends on gender, energy availability and dietary composition, and can be changed by meal timing (Hulver and Houmard 2003). It has been demonstrated that leptin concentration decreased after 10 weeks of food restriction-induced weight reduction and exercise in competitive body builders (Maestu et al. 2008). Increased training for two weeks resulted in decreased leptin concentrations after two hours rowing in male rowers (Ramson et al. 2008). Leptin administration results in increased spontaneous physical activity in animals (Hwa et al. 1997). There is a positive relationship between leptin concentration and physical activity particularly NEAT in humans (Franks et al. 2003).

It has been reported that leptin concentration does not change after short duration moderate or high intensity acute exercise (Kraemer et al. 1999; Fisher et al. 2001; Kyriazis et al. 2007). On the other hand, leptin concentration has been shown to



decrease immediately following cycling for two hours (Landt et al. 1997) and after maximum exercise in male rowers (Jurimae et al. 2007b). Additionally, running at 70% of  $\dot{V}O_2\text{max}$  leads to a decrease in fasting leptin concentration 48 hours following exercise but not immediately after exercise (Essig et al. 2000; Olive and Miller 2001). It has been suggested that altered leptin concentration might be linked to the energy expended during exercise (Hulver and Houmard 2003). High intensity, long duration exercise that leads to considerable energy expenditure might exert a more pronounced effect on leptin concentration.

Incremental and high intensity exercise (Bruce protocol) and cycling for 41 minutes in a fed state have been reported to cause an increase in postprandial leptin concentration (Sliwowski et al. 2001; Fisher et al. 2001), although one study showed a decrease in postprandial leptin concentration after running for two hours in a fed state (Duclos et al. 1999). Finally, there has been at least one report of no change in postprandial leptin concentration after moderate intensity exercise in fed state (Cheng et al. 2009). Clearly, the impact of exercise in a fed state on leptin concentration remains uncertain.

Adiponectin is secreted from adipose tissue and low levels of plasma adiponectin are associated with cardiovascular diseases and type II diabetes mellitus (Meier and Gressner 2004; Stanley et al. 2005). Adiponectin concentration in obese participants are lower than healthy participants (Arita et al. 1999). Adiponectin is increased after weight loss in diabetic and non-diabetic participants (Hotta et al. 2000). Central administration of adiponectin causes an increase in energy expenditure and a decrease in food intake in mice (Qi et al. 2004; Kubota et al. 2007). Peripheral administration of adiponectin leads to increased oxygen consumption and fat metabolism in mice (Yamauchi et al. 2001; Fruebis et al. 2001). There is no change in adiponectin concentration after exercise training for 24 weeks in sedentary and healthy participants (Hulver et al. 2002; Ryan et al. 2003; Yatagai et al. 2003). However, exercise combined with diet for 15 weeks leads to an increase in adiponectin concentration in obese men and women (Bruun et al. 2006). Several studies found there was no change in adiponectin concentration after acute exercise in healthy and obese participants (Ferguson et al. 2004; Jamurtas et al. 2006; Punyadeera et al. 2005). However, one study reported adiponectin concentration increased after running at 79%  $\dot{V}O_2\text{max}$  for

30 minutes but not after correcting for plasma volume changes in healthy men (Kraemer et al. 2003). It appears that there is no impact of acute exercise on adiponectin concentration.

Glucagon-like peptide 1 (GLP-1) is a hormone secreted from the L-cells of the small and large intestine, pancreas and also in the brain stem (Coll 2007; Huda et al. 2006; Stanley et al. 2005). Intravenous administration of GLP-1 leads to a decrease in food intake in lean and obese humans (Verdich et al. 2001). Postprandial GLP-1 has been reported to increase after incremental, moderate and high intensity exercise (Martin et al. 2007; Sliwowski et al. 2001; Ueda et al. 2009b). It has been observed that GLP-1 after exercise was lower in obese men than normal weight men (Ueda et al. 2009a). One study reported no difference in GLP-1 following either moderate or high intensity exercise (Ueda et al. 2009b).

Based on the discussion above and the information in Tables 1.5 and 1.6, it appears that exercise after a meal has the potential to change satiety hormones such as total PYY, PP, and GLP-1. However, in this situation, there appears to be no impact on the hunger hormone acylated ghrelin (or total ghrelin). On the other hand, exercise after an overnight fast has a powerful effect on acylated ghrelin but not total ghrelin concentration.

Subjects	Experiment design	Results	Reference
Well trained males (21 yrs)	Running at 72% $\dot{V}O_{2max}$ for 60 mins.	↓AUC of acylated ghrelin	Broom et al. 2007
Lean and obese	Cycling at 20 W and then increased by 20 W every 4 minutes until exhaustion	↓Acylated ghrelin obese > lean No change total ghrelin	Marzullo et al. 2008
Healthy males and females (24 yrs)	Cycling at LIE (50W) and HIE (100 W) for 30 mins.	No change in total ghrelin	Erdmann et al. 2007
Healthy males and females (25 yrs)	Running 73% $\dot{V}O_{2max}$ for 60 mins.	No change in total ghrelin	Burns et al. 2007
Elite males rowers (21 yrs)	Maximum 6000 m ergometer performance (~20 min).	↑Total ghrelin, ↓leptin	Jurimae et al. 2007b
Healthy males (23 yrs)	Resistance exercises (10 exercises, three circuits at 60% of 1 RM).	↓Total ghrelin, ↑ Total ghrelin 24 hrs	Ghanbari-Niaki 2006
Healthy males (young)	Running at 50%, 70% and 90% $\dot{V}O_{2max}$	No change in total ghrelin	Schmidt et al. 2004
Moderately trained males (21-44 yrs)	Running at 70% $\dot{V}O_{2max}$ (800 and 1500 kcal)	↓Leptin 48 hours 800, 1500 kcal	Essig et al. 2000
Healthy males (30 yrs)	Cycling for 2 hours at 75% $\dot{V}O_{2peak}$	No change in leptin	Landt et al. 1997
Trained males	Running at 70% $\dot{V}O_{2max}$ for 60 mins.	↓Leptin 48 hours	Olive and Miller 2001
Obese males (25 yrs)	Running at 50-60% $\dot{V}O_{2max}$ for 60 mins.	No change in leptin immediately, 24, 48	Kyriazis et al. 2007
Well trained males (21 yrs)	Running 70% $\dot{V}O_{2max}$ (60 mins), resistance exercise 90 mins (80% RM).	↓Acylated ghrelin running, resistance ↑ Total PYY running	Broom et al. 2009

Table 1.5 A summary of studies investigating the interaction between acute exercise and hormones in fasted state. PYY, peptide YY; PP, pancreatic polypeptide; GLP-1, glucagon like peptide 1. HIE, high intensity exercise. ↑, increase; ↓, decrease >, greater than; <, less than.

Subjects	Experiment design	Results	Reference
Moderately active males (25 yrs)	Exercise at 60% $\dot{V}O_2$ max for 50 mins before (BM) and after (AM) meal. Meal (M) only.	↑PYY <sub>3-36</sub> trend BM, AM > M No change in ghrelin, leptin	Cheng et al. 2009
Healthy males (19-24 yrs)	Resistance exercise with and without carbohydrate.	↑Total ghrelin	Ballard et al. 2009
Sedentary males (29 yrs)	Cycling for 41 mins after a meal.	↑Leptin 10%	Fisher et al. 2001
Runner men (41 yrs)	Running for 101 miles for 35 hrs (non-fasting).	↓Leptin	Landt et al. 1997
Runner males (41 yrs)	Running at 65-75% $\dot{V}O_2$ max for 2 hrs after breakfast	↓Leptin 2 hours after exercise.	Duclos et al. 1999
Healthy males and females (26 yrs)	Intermittent cycling 65% Maximum HR for 60 mins after consuming chocolate drink.	↑PP, ↑GLP, ↑Total PYY No change in total ghrelin	Martin et al. 2007
Obese, healthy men (22 yrs)	Constant cycling at 50% $\dot{V}O_2$ max for 60 mins after consumed breakfast.	↑ Total PYY ↑GLP-1 normal > obese	Ueda et al. 2009a
Healthy males (23 yrs)	Cycling at 75% and 50% $\dot{V}O_2$ max for 30 mins after consumed breakfast.	↑ PYY <sub>3-36</sub> HIE, LIE ↑GLP-1	Ueda et al. 2009b
Well trained males (21 yrs)	Running at 70% $\dot{V}O_2$ max (60 mins), resistance exercise 80% RM (90 mins) before test meal.	↑ Total PYY running No change in acylated ghrelin	Broom et al. 2009
Sedentary, overweight males (26 yrs) and females (23 yrs)	Four sessions exercise at 50-65% $\dot{V}O_2$ max (30% TEE) with (W) and without (WO) energy replacement. Ghrelin measured on the following day after test meal.	↑Acylated ghrelin females W, WO > B females AUC Total ghrelin W, WO > B	Hagobian et al. 2009

Table 1.6 A summary of studies investigating the interaction between acute exercise and hormones in fed state. PYY, peptide YY; PP, pancreatic polypeptide; GLP-1, glucagon like peptide 1. HIE, high intensity exercise. TEE, total energy expenditure. ↑, increase; ↓, decrease; >, greater than; <, less than. B, baseline. AUC, area under the curve. Age (means).

## **1.6 Conclusion**

Obesity is a major problem and increases the risk of many diseases such as various cancers and cardiovascular disease. Lack of physical activity and overconsumption causes an energy imbalance between energy expenditure and energy intake (Dishman et al. 2006). The most malleable component of energy output or energy expenditure is physical activity or exercise. Exercise can increase total energy expenditure directly and might also influence energy intake and energy expenditure indirectly through changing the concentration of hormones that are involved in energy balance (McMurray and Hackney 2005). Therefore, research into the effects of exercise on feeding behaviour and energy balance are very important in order to understand the role of exercise in the prevention of obesity and obesity-related diseases. The impact of acute exercise on energy balance is associated with several factors such as mode, intensity and duration of exercise. Additionally, participant status such as physical activity level and dieting are very important as these might influence energy balance. Nevertheless, at the present time, the impact of a single bout of exercise on post-exercise energy intake is still rather controversial with inconsistent findings from previous research. Little is known about the impact of a single bout of exercise on physical activity energy expenditure. Therefore, the overarching aim of the work described in this thesis was to investigate the impact of a single bout of exercise on post-exercise energy intake and physical activity energy expenditure. It is proposed that such changes may be secondary to changes in circulating energy balance hormones such as acylated ghrelin, leptin, PYY, PP and adiponectin.

## CHAPTER 2

### GENERAL METHODS

#### 2.1 Introduction

The experiments described in this thesis took place in the Applied Physiology laboratory at the University of Bath. Males aged between 18 and 45 years participated in each study. In order to assess eligibility, volunteers completed a health history questionnaire (Appendix 1) and a Physical Activity Readiness Questionnaire (PAR-Q). The participants were non smokers with a body mass index of less than 30 kg/m<sup>2</sup> who were not on medication. Furthermore, their body mass had not changed by more than 5 kg in the last six months.

#### 2.2 Preliminary tests

Anthropometry, blood pressure, resting heart rate, sub-maximal oxygen consumption, maximal oxygen consumption ( $\dot{V}O_{2\max}$ ) and body composition were assessed at least seven days before each main trial.

##### 2.2.1 Anthropometry

Height, weight, waist and hip circumference were measured. Height was assessed using a fixed stadiometer (Holtain Limited, United Kingdom). Body weight was measured using an Avery balance scale (Weylux, United Kingdom). Body mass index (BMI) was calculated using the ratio between weight and height in square metres. Waist and hip ratio was determined by the narrowest part of the waist and the widest part of the hip.

##### 2.2.2 Body composition

Total body fat, lean and bone mineral content of all participants were assessed by using dual-energy X-ray absorptiometry (Hologic Inc, USA). The basic principle is based on

the different bone and soft tissue attenuation characteristics at the two pulsed X-ray levels. The raw scan data, containing the attenuation values for tissue, bone, and the calibration drum were captured and transferred to a computer programme. An algorithm interprets each pixel, creates an image and quantitative measurement of the bone and body tissue. Participants were placed in a supine position with arms and legs close to their body for a whole body scan following the manufacturer's recommended protocol. Whole body and regional lean mass (muscle mass and non-fat soft tissue), bone and fat mass were determined using the different bone and soft tissue attenuation characteristics at the two pulsed X-ray levels and manufacturer's algorithm. The whole body scan took from 10 to 15 minutes and participants did not need to fast beforehand.

Body composition was measured at the University of Bath except in Chapter 3 where this was measured at the Royal National Hospital for Rheumatic Diseases, Bath (RNHRD).

### **2.2.3 Sub-maximal oxygen consumption**

The speed of moderate (40%  $\dot{V}O_{2max}$ ) and high (70%  $\dot{V}O_{2max}$ ) intensity exercise in the main trials were determined by the relationship between speed and oxygen consumption of the sub-maximal treadmill test (Woodway ELG70, Germany). The speed of the walking test started from 4-7 km/h and in the running test from 7-10 km/h (Chapters 3 and 4). Additionally, walking speed started at 5-8 km/h (Chapter 5) whereas the running speed started at 8-11 km/h (Chapters 5 and 6). The duration of each test was 16 minutes and was composed of four stages. Expired gas, heart rate (Polar, Finland) and rating of perceived exertion scale (Borg 1973) were collected for one minute in all four stages (Appendix 6).

### **2.2.4 Maximum oxygen consumption**

The linear regression equation between heart rate and speed from the sub-maximal oxygen consumption test was used to determine the speed for the maximal oxygen consumption ( $\dot{V}O_{2max}$ ) test. Maximal oxygen consumption was determined during an incremental running test where the gradient increased by 2.5% every three minutes from

an initial 3.5% until fatigue. Expired gas samples, heart rate and rating of perceived exertion were collected in each stage and at maximum.

Oxygen (O<sub>2</sub>) and carbon dioxide (CO<sub>2</sub>) concentrations were measured using a gas analyser (Series 1400, Servomex Ltd., Sussex, United Kingdom). Gas analysers were calibrated within the physiological range within one hour of each test (British Oxygen Company, United Kingdom). The volume of expired gas was measured using a dry gas meter (Harvard Apparatus., Kent, United Kingdom) and corrected to standard temperature and pressure. Room temperature and barometric pressure were measured. Oxygen consumption was calculated for each expired air sample.

### **2.3 Energy intake**

A buffet meal was provided for 30 minutes on each main trial day following rest or exercise trials in all experiments except the first experiment (Chapter 3). The buffet meal was designed to be similar to typical lunchtime foods (Table 2.1). Food and fluid were weighed and recorded before and after eating (Appendix 5). Additionally, participants weighed and recorded their daily food and fluid intake on the main trial day and for two days after each main trial. Dietary composition was analysed using computer software (COMP-EAT version 5, Carlson Bengston Consultants Limited, London).



---

**Buffet meal**

---

White bread  
Brown bread

Butter  
Strawberry jam  
Orange marmalade  
Honey  
Mayonnaise  
Nutella  
Cheddar cheese

Sliced ham  
Tuna

Sliced lettuce  
Sliced tomato  
Sliced carrots  
Apple  
Orange  
Banana

Orange juice  
Coca-cola  
Water  
Coffee  
Tea  
Milk  
Sugar

Chocolate ice-cream  
Vanilla ice-cream

Kit-Kat  
Plain potato crisps  
Chocolate cookies

---

Table 2.1 Food items provided during a buffet meal in Chapters 4, 5 and 6. Adapted from a previous study (Pomerleau et al. 2004).

The compensation to exercise is defined as the energy intake during the buffet meal expressed relative to energy expenditure and taking into account energy intake during the rest trial (Pomerleau et al. 2004). Table 2.2 shows an example of the compensation to exercise-induced energy expenditure during a buffet meal.  $\text{Compensation} = [(\text{energy intake during the buffet meal in each exercise trial} - \text{energy intake in control rest})]$

trial)/energy cost of exercise above resting energy expenditure)] x 100. For example, a value of 0% represents energy intake after exercise being equivalent to energy intake after the rest trial; a value of 100% represents energy intake after exercise being 100% greater than after rest and the difference of energy intake between rest and exercise is equivalent to the energy expended in the exercise session above rest; a value of -100% represents energy intake after rest being 100% greater than after exercise and the difference of energy intake between rest and exercise is equivalent to the energy expended in the exercise session above rest.

	EE Rest	EE Exercise	EI Rest	EI Exercise
150 %	100	900	2000	3200
100 %	100	900	2000	2800
50 %	100	900	2000	2400
0 %	100	900	2000	2000
-50 %	100	900	2000	1600
-100 %	100	900	2000	1200
-150 %	100	900	2000	800

Table 2.2 Example calculation of compensation to exercise-induced energy expenditure during a buffet meal. EI, energy intake; EE, energy expenditure.

## 2.4 Subjective feelings of hunger and fullness

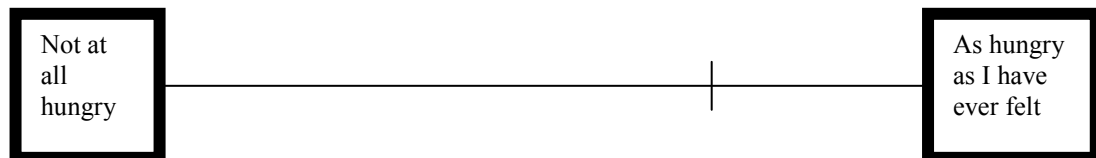
100 mm visual analogue scales (VAS) were used to assess subjective feelings of hunger and fullness in all experiments (Chapters 3, 4, 5 and 6). There are four questions to assess subjective feelings of hunger and fullness rating including “how hungry do you feel?”, “how full do you feel?”, “how strong is your desire to eat” and “how strong is your urge to eat” (Hill et al.1987). The VAS is a straight line with two anchors at each end. The distance between anchors was 100 mm with “Not at all hungry” and “As hungry as I have ever felt” on the left and right side. Participants indicated their appetite by marking on the line (Figure 2.1). Subjective feelings of hunger and fullness were determined on the main trial days before, after exercise or rest and after eating (Chapters 3, 4, 5 and 6). In addition, subjective appetite ratings were also assessed starting from 12.00 a.m. to 6 p.m. (Chapters 4 and 5). Timed watches were provided in

order to remind participants to complete this measure every 30 minutes. In Chapter 6, subjective feelings of hunger and fullness were assessed prior to food restriction.

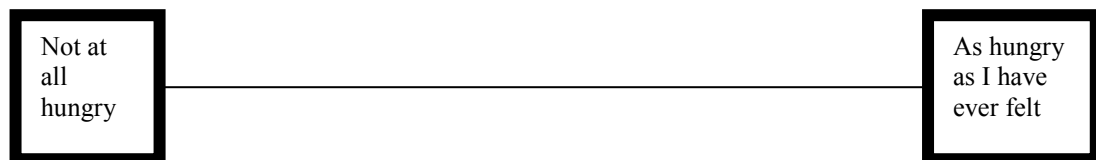
**Instructions:** Please mark on the line

**Example:**

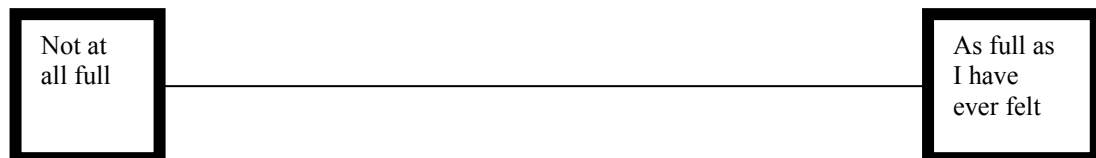
**Time:** 12.30 A.M. / ~~P.M.~~ / -before/after food or snack or drink



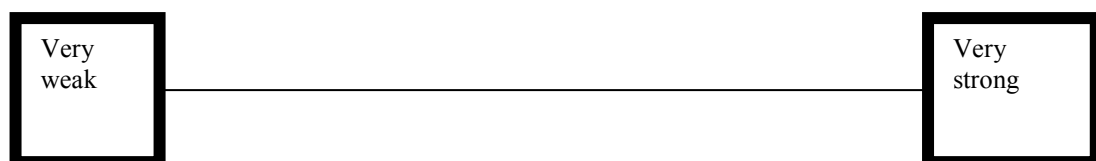
**1. How hungry do you feel?**



**2. How full do you feel?**



**3. How strong is your desire to eat?**



**4. How strong is your urge to eat?**

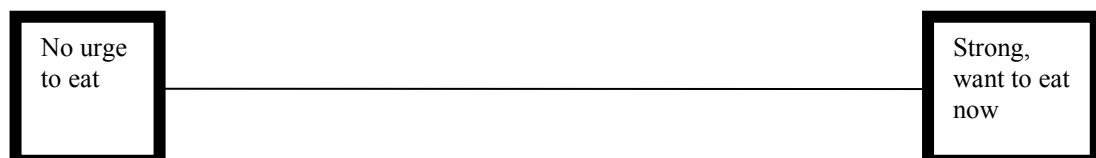


Figure 2.1 100 mm visual analogue scales (Hill et al. 1987).

## 2.5 Energy expenditure during rest or exercise in the laboratory

Energy expenditure in the laboratory was determined by indirect calorimetry. Gas analysers were calibrated within the physiological range within one hour of each trial (British Oxygen Company, United Kingdom). Expired gas was collected for one minute every ten minutes in each exercise trial and for five minutes in rest trials in order to estimate energy expenditure. Oxygen (O<sub>2</sub>) and carbon (CO<sub>2</sub>) dioxide concentrations were measured using a paramagnetic O<sub>2</sub> gas analyser and an infrared CO<sub>2</sub> gas analyser (Series 1400, Servomex Ltd., Sussex, United Kingdom). Expired air volume was measured using a dry gas meter (Harvard Apparatus, Kent, United Kingdom). Carbohydrate and fat are the major energy substrates and during exercise protein oxidation provides only one to ten percent of energy expenditure (Jequier et al. 1987). Carbohydrate and lipid oxidation were calculated in order to estimate energy expenditure (Jequier et al. 1987). Oxidising one gram of carbohydrate uses 0.828 O<sub>2</sub> and assumed to produces 0.828 CO<sub>2</sub> and 17 kJ whereas oxidising one gram of lipid uses 1.989 O<sub>2</sub> and assumed to produces 1.419 CO<sub>2</sub> and 39 kJ

$$\text{Total O}_2 \text{ consumption} = 0.828 \text{ carbohydrate} + 1.989 \text{ fat}$$

$$\text{Total CO}_2 \text{ production} = 0.828 \text{ carbohydrate} + 1.419 \text{ fat}$$

## 2.6 Physical activity energy expenditure

Participants wore a physical activity monitor (Actiheart, Cambridge Neurotechnology Ltd. Cambridge) in order to estimate physical activity energy expenditure outside the laboratory. This physical activity monitor estimates physical activity energy expenditure in free-living activity using combined movement and heart rate variability sensing (Brage et al. 2004). The physical activity monitors were applied on each participant's chest on either the third inter-costal space or below the apex of sternum (Figure 2.2). Previous work has shown that either placement give the same predicted of physical activity energy expenditure (Thompson et al. 2006). The epoch (i.e. subdivision of a period) length for recording was one minute. Daily total energy expenditure was the sum of physical activity energy expenditure (PAEE), diet induced thermogenesis (DIT), and resting energy expenditure (REE). Resting energy

expenditure was estimated from Schofield equation (Schofield 1985) while diet-induced thermogenesis was assumed to equal 10% of total energy expenditure. PAEE was estimated using a Branched equation (Brage et al. 2004). PAEE and time spent in classified metabolic equivalent (METs) zones (Pollock et al. 1998) such as very light (1.1-2.39 METs), light (2.4-4.79 METs), moderate (4.8-7.19 METs), hard (7.2-10.19 METs), and very hard ( $\geq 10.2$  METs) were analysed from Actiheart Software version 2.170 as previously described (Lund 2009; Thompson et al. 2006).

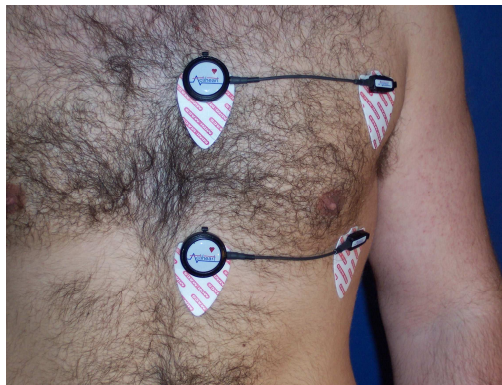


Figure 2.2 Physical activity monitor. This image shows two instruments in different locations.

## 2.7 Blood sampling and assays

Participants reported to the laboratory in the morning after an overnight fast (Chapters 3, 4, 5 and 6). Venepuncture was used in all experiments (Chapters 3, 4, 5 and 6) whilst cannulation was used in the final study (Chapter 6). Participants remained in the supine position (Chapter 3), sitting (Chapter 6) and standing position blood sampling (Chapters 4 and 5).

Venous blood samples were taken from a forearm vein and collected into the anti-coagulant ethylenediaminetetra tubes (EDTA) and serum-separator tubes (allowed to clot for 15 minutes at room temperature) before rest or exercise after an overnight fast (no food or drink except water for at least ten hours). The tubes were spun at 5000 rpm

at 5°C for ten minutes. Serum and plasma were removed and the sample stored at -70°C for analysis.

Additionally, blood samples were collected in tubes containing EDTA and p-hydroxymercuribenzoic acid to prevent the degradation of acylated ghrelin by proteases. Samples were centrifuged at 5000 rpm for ten minutes at 4°C and then, supernatants were transferred to separate tubes. 100µL of 1N HCL per ml was added and samples centrifuged at 5000 rpm for five minutes at 4°C (Spi-bio, France). Supernatants were transferred to separate tubes and stored at -70°C prior to analysis.

Whole blood in EDTA tubes was used to measure the concentration of glucose and lactate (YSI 2300 STAT Plus, Yellow Springs, United State) and the white blood cell count by an automated haematology analyser (Sysmex SF 2300, United Kingdom). Plasma acylated ghrelin (Chapters 3, 4, 5 and 6) was measured using Enzyme-linked immunoassay (ELISA; Spi-bio, France). Serum leptin (Chapters 3, 4, 5 and 6) and adiponectin (Chapter 4) were measured using ELISA (R&D Systems, Abingdon, United Kingdom). Plasma total peptide YY (Chapters 5 and 6) and pancreatic polypeptide (Chapter 6) were measured using ELISA (LINCO research, United State). Plasma triglyceride (RANDOX laboratories Ltd., Ardmore, United Kingdom) and plasma free fatty acids (NEFA-C, Wako Chemical GmbH, Germany) were measured using enzymatic colorimetric method on a Roche Cobas Mira (Switzerland). Orexin A (Chapter 4) was measured using radioimmunoassay (Phoenix Pharmaceuticals, United State) and samples were sent to Germany for analysis (Appendix 9). The reported manufacturer intra-assay precision coefficient of variation (CV) for assays were as follows: acylated ghrelin 3.6%, leptin 3.2%, adiponectin 3.5%, total PYY 2.3% and PP 4.3%. Table 2.3 shows measured coefficient of variation (CV) for acylated ghrelin, leptin, adiponectin, total PYY and PP concentrations.

Assays	CV (%)	Number
Acylated ghrelin	3.1	43
Leptin	3.1	43
Adiponectin	2.8	10
Total PYY	3.5	21
PP	2.4	11

Table 2.3 Coefficient of variation (CV) for acylated ghrelin, leptin, adiponectin, total PYY and PP.

## CHAPTER 3

### THE EFFECT OF EXERCISE AT 40% AND 70% $\dot{V}O_{2\max}$ ON ENERGY INTAKE, PHYSICAL ACTIVITY ENERGY EXPENDITURE AND ENERGY BALANCE HORMONES IN SEDENTARY MALES AGED BETWEEN 18 AND 34 YEARS

#### 3.1 Introduction

Obesity is strongly implicated in several diseases (Haslam and James 2005) and is caused by an imbalance between energy intake and energy expenditure. Exercise is one component of energy expenditure and this might influence post-exercise energy balance via change in energy intake and physical activity energy expenditure possibly via changes in energy balance hormones such as ghrelin and leptin.

The impact of acute exercise on subsequent energy expenditure has been previously studied. McLaughlin et al. (2006) found that there was no change in physical activity energy expenditure assessed using a heart rate monitor and physical activity records for eight days during which participants completed four sessions of moderate intensity exercise. Interestingly, one study demonstrated that moderate intensity exercise induced an increase in energy expenditure estimated using a heart rate monitor on the following day while high intensity exercise led to a decrease in energy expenditure in obese boys (Kriemler et al. 1999). However, the energy expended during high intensity exercise was greater than during moderate intensity exercise (Kriemler et al. 1999). It is unclear if post-exercise physical activity energy expenditure in free living conditions would have been different if energy expenditure in both exercise trials (moderate / high intensity) had been equivalent (Kriemler et al. 1999).

The influence of exercise intensity on subsequent energy intake has been investigated but the results are still not clear. For example, Pomerleau et al. (2004) found that energy intake during a buffet meal increased after high intensity exercise; however, Kissileff et al. (1990) reported that energy intake decreased after highly intensive exercise. In addition, some studies have demonstrated that energy intake and sensations



of hunger do not change after moderate intensity exercise (Imbeault et al. 1997; Pomerleau et al. 2004).

There are several hormones that influence energy intake and physical activity energy expenditure (e.g., ghrelin and leptin). Leptin infusion results in increased energy expenditure in animals (Hwa et al. 1997). Several studies have investigated the impact of exercise on leptin concentrations. Some studies reported that there was no change in leptin concentration immediately after high intensity exercise (Oliver and Miller 2001) and moderate intensity exercise (Kyriazis et al. 2007). Another study demonstrated that leptin concentration decreased two hours after exercise (Landt et al. 1997). It has been demonstrated that administration of ghrelin leads to increased food intake and decreased spontaneous locomotor activity in rats (Tang-Christensen et al. 2004). Additionally, it has been reported that administration of total ghrelin results in an increase in energy intake in humans (Wren et al. 2001). Several researchers have studied the influence of acute exercise on plasma total ghrelin concentration and it was found that plasma concentration of total ghrelin did not change after moderate incremental exercise (Zoladz et al. 2005), intermittent treadmill exercise (Kraemer et al. 2004) and graded treadmill exercise at 50%, 70% and 90%  $\dot{V}O_2\text{max}$  (Schmidt et al. 2004). However, only a few researchers have investigated the effect of acute exercise on acylated ghrelin and it was found that plasma acylated ghrelin decreased after high intensity exercise in well trained men (Broom et al. 2007; Broom et al. 2009). Little is known about the impact of a single bout of exercise at moderate and high intensity on acylated ghrelin and leptin concentrations in sedentary men.

Therefore, the main purpose of this study was to investigate the impact of an acute bout of exercise at moderate (40%  $\dot{V}O_2\text{max}$ ) and high intensity (70%  $\dot{V}O_2\text{max}$ ) on post-exercise energy intake, appetite, physical activity energy expenditure and energy balance hormones in young sedentary men. The main hypothesis is that high intensity exercise might decrease post-exercise energy intake, circulating leptin and plasma acylated ghrelin concentration in comparison to moderate intensity exercise.

## 3.2 Methods

### 3.2.1 Participant recruitment and eligibility

	Mean±SE
Age (years)	26±1
Body mass (kg)	70.6±2.7
Height (cm)	170±2.0
BMI (kg/m <sup>2</sup> )	23±1
Waist (cm)	78±2.9
Hip (cm)	94.3±2.7
Waist/Hip ratio	0.8±0
Total percent body fat (%)	21±1
Total fat mass (kg)	15±1
Total lean mass (kg)	55±2
Blood pressure: systolic/diastolic (mmHg)	121±3 /78±2
Resting heart rate (beats /min)	67±2
$\dot{V}O_2\text{max}$ (ml/kg/min)	50.4±2.9

Table 3.1 Participant characteristics. Values are means±SE (n=12). BMI, body mass index;  $\dot{V}O_2\text{max}$ , maximal oxygen consumption. Body composition determined by dual-energy x-ray absorptiometry (DEXA).

Twelve sedentary males aged between 18 and 34 years participated in this study, which had approval from the Local NHS Ethics Committee (Table 3.1). In order to assess eligibility, participants completed a health history questionnaire and a Physical Activity Readiness Questionnaire (PAR-Q). The participants were non smokers with a body mass index of less than 30 kg/m<sup>2</sup>. Furthermore, their body weight had not changed more than 5 kg in the last six months. In addition, to be eligible to take part, they had to confirm that they did not do any moderate intensity activity more than 30 minutes per day and no more than three times per week (from self report, Appendix 1). The participants provided written informed consent.

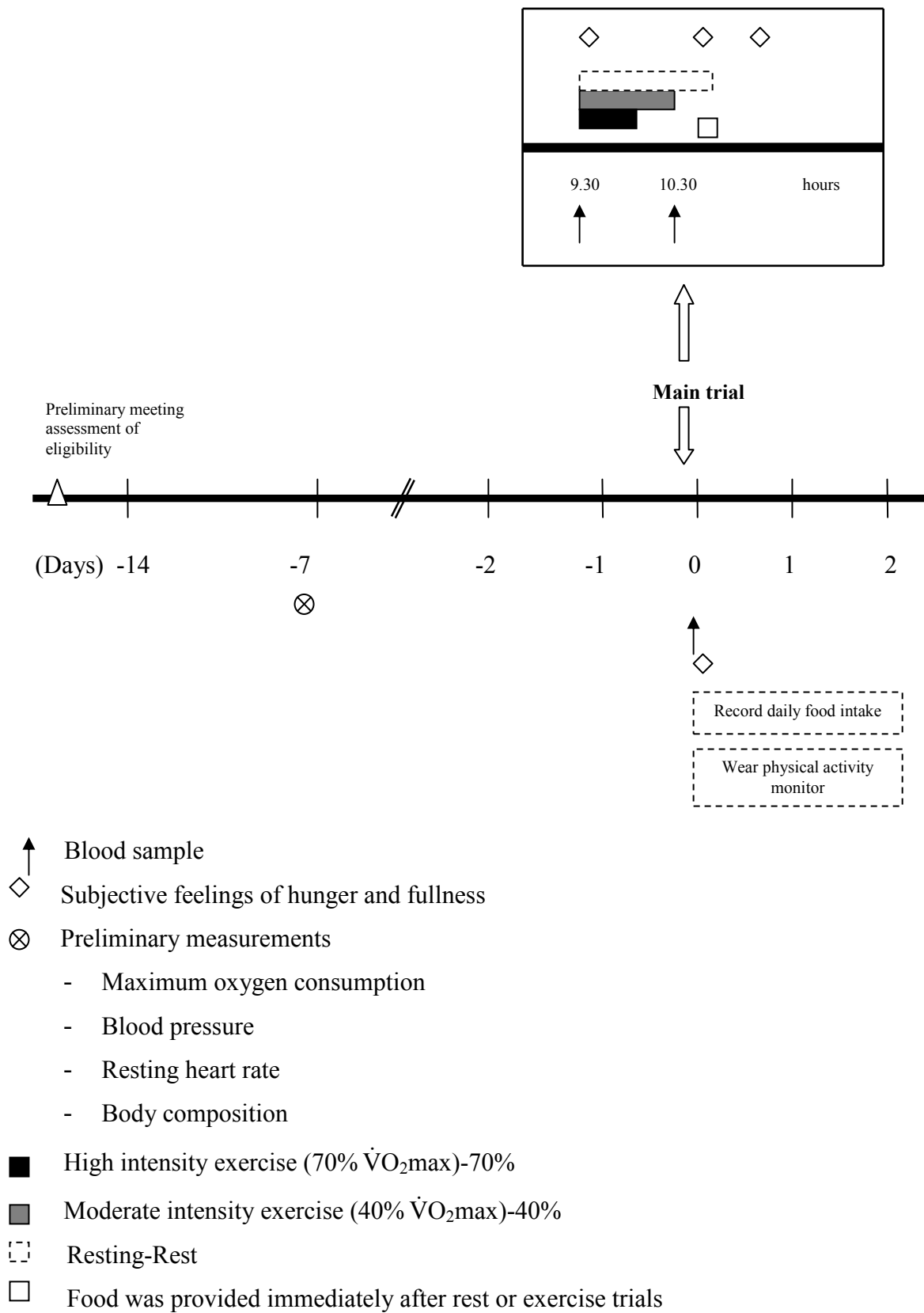


Figure 3.1 Study protocol. Main trial represents either rest 40%  $\dot{V}O_2\text{max}$  or 70%  $\dot{V}O_2\text{max}$  in a randomized order with at least one week between trials.

### **3.2.2 Experimental design**

On two occasions participants performed a standardised bout of exercise and on another occasion they rested in the laboratory for 80 minutes, each trial separated by at least one week (Figure 3.1). In the low intensity exercise trial, participants walked on a treadmill at a target exercise intensity of 40%  $\dot{V}O_{2\max}$  until they had expended 6 kcal/kg (~60 minutes). In the high intensity exercise trial, participants expended the same amount of energy, but at an intensity of 70%  $\dot{V}O_{2\max}$  (~40 minutes). This model was based on the previous study of Pomerleau et al. (2004).

### **3.2.3 Energy intake**

Food (rice cakes and nutella) was provided on the main trial day immediately after the rest or exercise trials to examine immediate post-exercise/rest energy intake. Additionally, energy intake outside the laboratory was assessed. To do this, participants weighed and recorded their daily food intake on the main trial day and for two days after each main trial (General Methods).

### **3.2.4 Subjective feelings of hunger and fullness**

As explained in the general methods (Chapter 2) subjective feelings of hunger and fullness (Appendix 3) were assessed using 100 mm visual analogue scales (VAS). These were assessed before and after exercise as well as after eating on each main trial day.

### **3.2.5 Energy expenditure and physical activity energy expenditure**

Expired gas was collected for one minute every ten minutes for each exercise trial and for five minutes in the rest trial in order to estimate energy expenditure using indirect calorimetry. Further details on these measurements can be found in the general methods (Chapter 2). To examine physical activity energy expenditure outside the laboratory, participants wore a physical activity monitor (Actiheart, Cambridge Neurotechnology

Ltd., Cambridge) on the main trial day and also two days following each main trial (Chapter 2).

### **3.2.6 Energy intake and physical activity energy expenditure control**

Participants were asked to replicate their food and fluid intake for two days before each main trial day. Participants were also asked to avoid alcohol and caffeine for 48 hours before each main trial.

### **3.2.7 Statistical analysis**

Sample size was estimated using previous work that shows that energy intake at the first meal following acute exercise is 130 kcal greater than a control trial where the standard deviation of the difference is 170 kcal (Pomerleau et al. 2004). Therefore, to detect a mean difference of 130 kcal in the present trial requires a estimated sample size of 14 and allowing for two participants dropout (80% power and 5% alpha).

All data were analysed using Statistical software (SPSS 14.0, United States). Two-way repeated measures ANOVA and *t*-tests were used to analyse differences in leptin, acylated ghrelin, blood glucose, blood lactate concentrations and subjective feelings of hunger and fullness. Paired *t*-tests were used for *post hoc* comparisons with a Bonferroni method. Statistical significance was accepted at the 5% level ( $P < 0.05$ ). Data are presented as means  $\pm$  SE.

### 3.3 Results

#### 3.3.1 Trial data

	Rest	40%	70%
$\dot{V}O_2$ (ml/kg/min)	3.8±0.2	20.4±1.1 <sup>a</sup>	34.6±1.5 <sup>b,c</sup>
Time (min)	80±0	62±3 <sup>a</sup>	39±1 <sup>b,c</sup>
Energy expenditure (kcal)	106±7	430±17 <sup>a</sup>	432±17 <sup>b</sup>
Total carbohydrate oxidation (g)	9.2±2.2	61.1±5.1 <sup>a</sup>	80.3±6.7 <sup>b</sup>
Total fat oxidation (g)	7.5±0.5	19.7±2.1 <sup>a</sup>	11.5±2.3
Water intake (ml)	139±73	195±48	320±141
Body weight change (kg)	0.2±0	0.3±0 <sup>a</sup>	0.4±0 <sup>b</sup>

Table 3.2 Trial data in rest and exercise trials. Values are means±SE (n=12).  $\dot{V}O_2$ , average oxygen consumption every ten minutes; water intake, during rest or exercise trials; body weight change, body weight before and after rest or exercise. 40%, exercise at 40%  $\dot{V}O_{2max}$ ; 70%, exercise at 70%  $\dot{V}O_{2max}$ . <sup>a</sup>, significantly different between rest and exercise at 40%  $\dot{V}O_{2max}$ ; <sup>b</sup>, significantly different between rest and exercise at 70%  $\dot{V}O_{2max}$ ; <sup>c</sup>, significantly different between exercise at 40%  $\dot{V}O_{2max}$  and 70%  $\dot{V}O_{2max}$  ( $P<0.05$ ).

As shown in Table 3.2, energy expenditure was similar in both exercise trials. This was achieved through the manipulation of exercise intensity and duration. The mean oxygen uptake during exercise was 20.4 (40%  $\dot{V}O_{2max}$ ) and 34.6 (70%  $\dot{V}O_{2max}$ ) ml/kg /min respectively (Student's *t*-test, all  $P<0.01$ ). The mean time of exercise was 62 minutes at 40%  $\dot{V}O_{2max}$  and 39 minutes at 70%  $\dot{V}O_{2max}$  (all  $P<0.01$ ).

### 3.3.2 Energy intake

Energy intake (kcal)	Rest	40%	70%
After trial (kcal)	248±44	266±30	290±50

Table 3.3 Energy intake immediately after each main trial. Values are means±SE (n=12). After each trial, nutella and rice cakes were provided. 40%, exercise at 40%  $\dot{V}O_2$ max; 70%, exercise at 70%  $\dot{V}O_2$ max.

Table 3.3 presents energy intake immediately after rest or exercise trials. There was no statistically significant difference in absolute energy intake between trials.

## 3.3.3 Subjective feelings of hunger and fullness

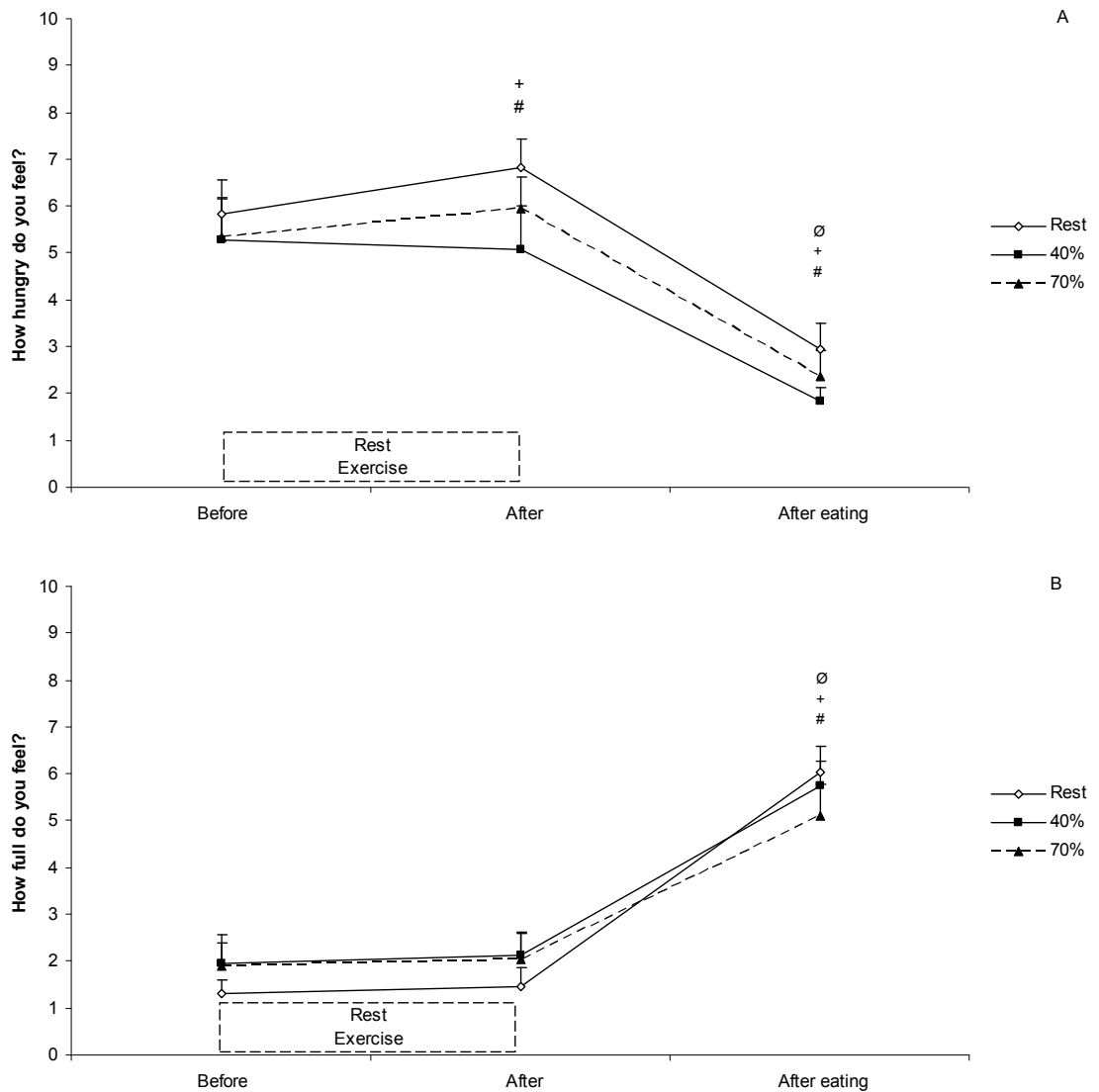


Figure 3.2 Subjective feelings of hunger and fullness using a 100 mm visual analogue scale (means $\pm$ SE) before and after rest or exercise and also immediately after eating (n=12). A, how hungry do you feel; B, how full do you feel. Data have been converted to a 0-10 scale for presentation. 40%, exercise at 40%  $\dot{V}O_2$ max; 70%, exercise at 70%  $\dot{V}O_2$ max. #, significantly different between before and after (rest or eating) in rest trials; +, significantly different between before and after (exercise or eating) in exercise at 40%  $\dot{V}O_2$ max; Ø, significantly different between before and after (exercise or eating) in exercise at 70%  $\dot{V}O_2$ max ( $P<0.05$ ).



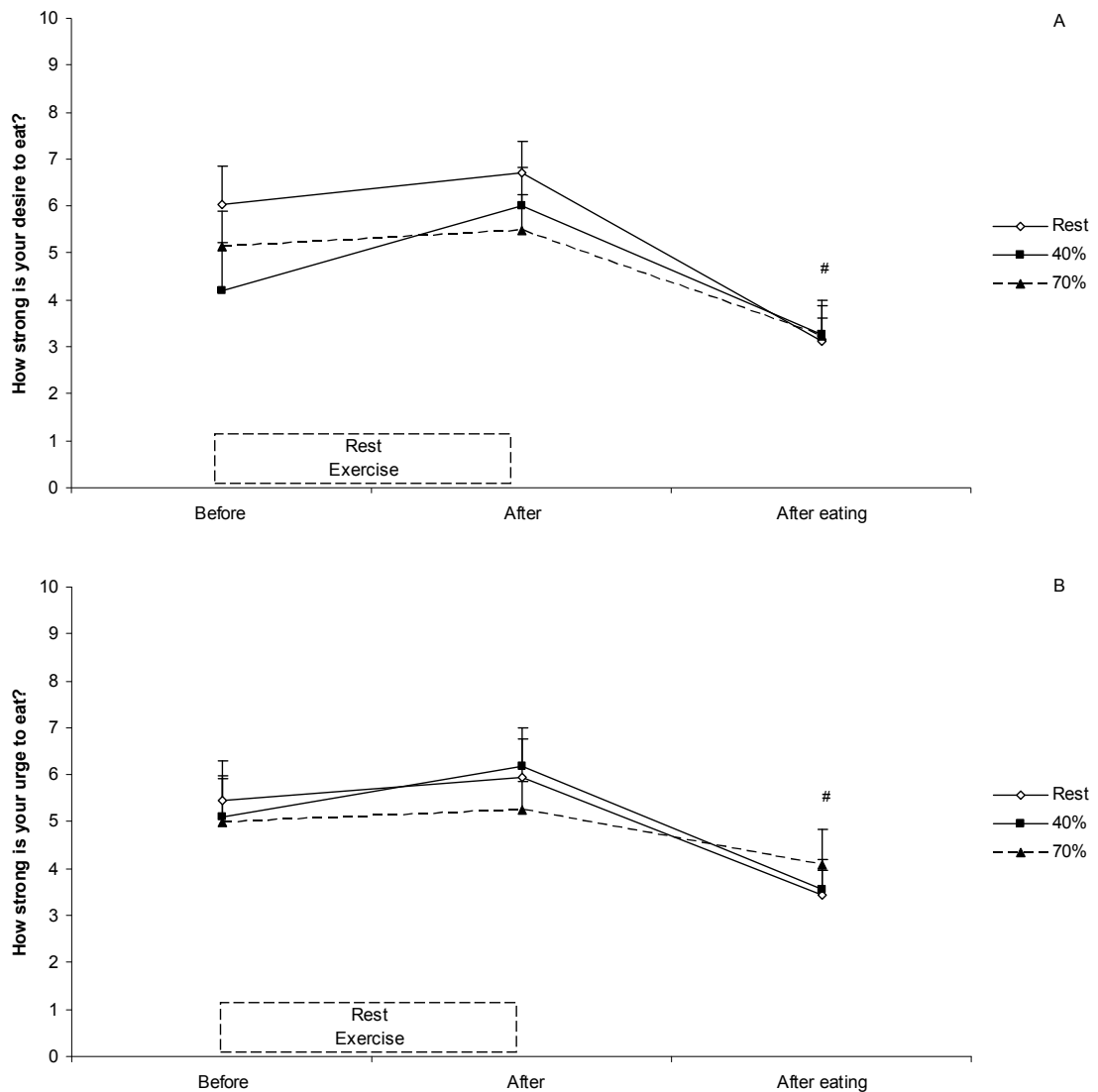


Figure 3.3 Subjective feelings of desire and urge to eat using a 100 mm visual analogue scale (means±SE) before and after rest or exercise and also immediately after eating (n=12). A, how strong is your desire to eat; B, how strong is your urge to eat. Data have been converted to a 0-10 scale for presentation. 40%, exercise at 40%  $\dot{V}O_2$ max; 70%, exercise at 70%  $\dot{V}O_2$ max. <sup>#</sup>, significantly different between before and after (rest or eating) in rest trials ( $P < 0.05$ ).

Subjective feelings of hunger and fullness were assessed before and immediately after rest or exercise and also after eating rice cakes and nutella. According to Figures 3.2 and 3.3, it can be seen that there was no significant difference in subjective feelings of hunger and fullness between trials. There was a time effect for hunger, fullness, desire and urge to eat, as a result of consumption of a meal ( $P < 0.01$ ).

### 3.3.4 Physical activity energy expenditure

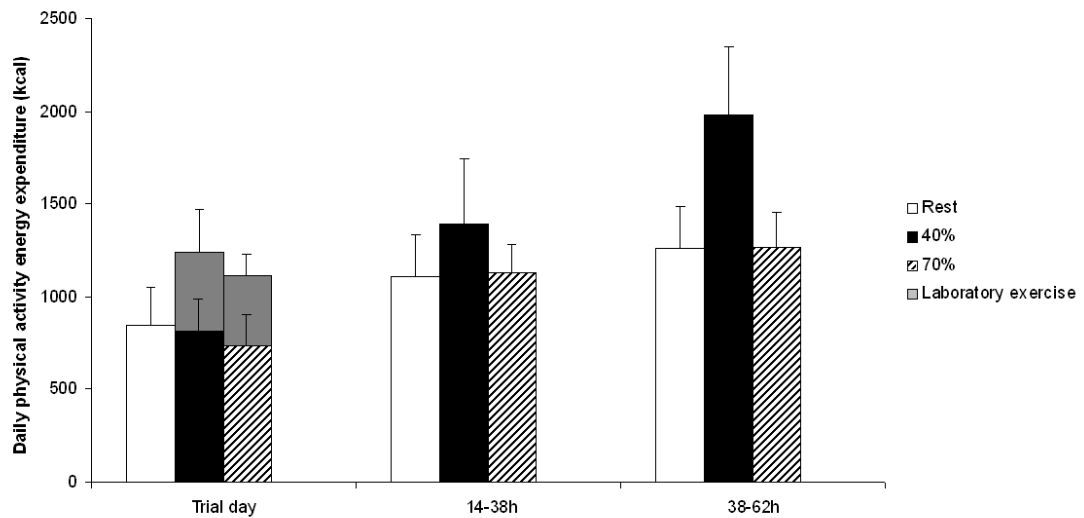


Figure 3.4 Physical activity energy expenditure (n=6). Values are means  $\pm$  SE. Physical activity energy expenditure (PAEE) on the trial day, PAEE after subtraction of energy expended during rest or exercise in the laboratory; 14-38h, between 14-38 hours; 38-62h, between 38-62 hours after main trials. 40%, exercise at 40%  $\dot{V}O_{2max}$ ; 70%, exercise at 70%  $\dot{V}O_{2max}$ .

Figure 3.4 shows physical activity energy expenditure on each trial day, between 14-38 hours and 38-62 hours after each trial. Unfortunately, there were problems with the physical activity monitors for some individuals and so these results are n=6. Two-way ANOVA revealed a trend for the interaction between trial and time for PAEE after subtraction of exercise in the laboratory ( $P=0.09$ ). Further analysis of these data showed that physical activity energy expenditure between 38 and 62 hours after exercise at 40%  $\dot{V}O_{2max}$  was greater than rest ( $P=0.05$ ).

### 3.3.5 Blood parameters

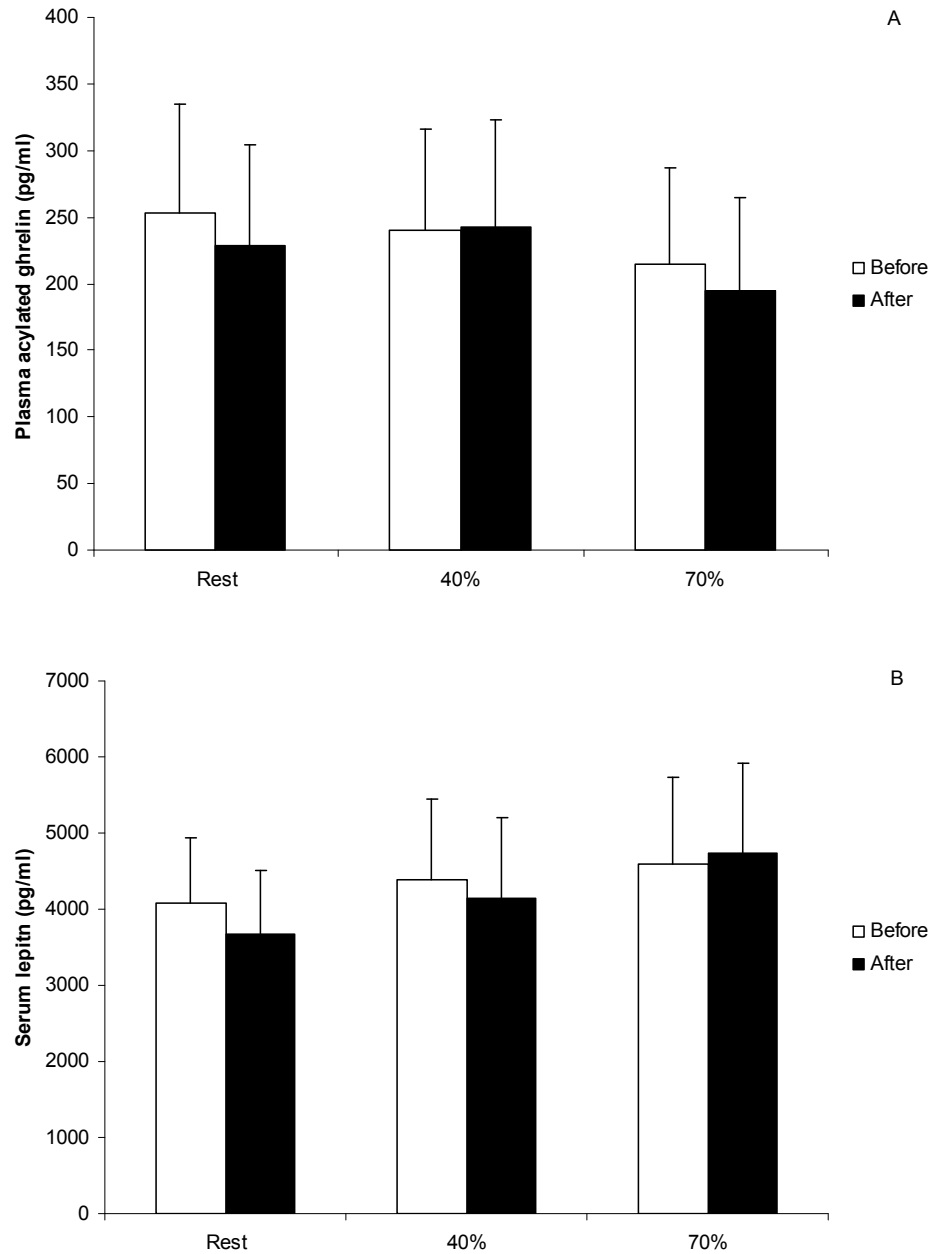


Figure 3.5 Plasma acylated ghrelin(A) and serum leptin (B) concentrations before and immediately after rest and exercise trials (n=12). Values are means  $\pm$  SE. 40%, exercise at 40%  $\dot{V}O_2\text{max}$ ; 70%, exercise at 70%  $\dot{V}O_2\text{max}$ .

Figure 3.5 shows concentrations of acylated ghrelin and leptin before and after exercise or rest. There was no difference in plasma acylated ghrelin and serum leptin concentrations between rest and exercise trials.

	Rest	40%	70%
Blood glucose (mmol/L )			
- Before	4.3±0.1	4.5±0.1	4.3±0.1
- After	4.1±0.1	4.3±0.1	4.3±0.1
Blood lactate (mmol/L)			
- Before	0.6±0.1	0.6±0.1	0.7±0.1
- After	0.5±0	0.5±0	1.6±0.2 <sup>b</sup>

Table 3.4 Blood glucose and blood lactate during rest and exercise trials. Values are means±SE (n=12). 40%, exercise at 40%  $\dot{V}O_{2max}$ ; 70%, exercise at 70%  $\dot{V}O_{2max}$ . <sup>b</sup>, significantly different between rest and exercise at 70%  $\dot{V}O_{2max}$ .

Table 3.4 shows blood glucose and blood lactate concentrations before and after exercise. Two-way ANOVA showed a trial and time interaction ( $P < 0.01$ ) for lactate concentration. There was no significant difference in blood glucose concentration after rest and exercise trials (Table 3.4).

### 3.4 Discussion

This study aimed to investigate the impact of a single bout of exercise (at 40% and 70% of  $\dot{V}O_2\text{max}$ ) on energy intake, physical activity energy expenditure (PAEE) and energy balance hormone concentrations in young sedentary males. Interestingly, there was a trend for PAEE to increase between 38 and 62 hours following moderate intensity exercise. However, there was no change in immediate post-exercise energy intake immediately after exercise and leptin and acylated ghrelin concentrations were not affected by moderate or high intensity exercise.

The main finding of this study was the trend for daily PAEE to increase between 38 and 62 hours following moderate intensity exercise at 40%  $\dot{V}O_2\text{max}$ . Kriemler et al. (1999) demonstrated that energy expenditure between 1.00 and 7.00 pm estimated using heart rate on the day after moderate intensity exercise was greater than after high intensity exercise but was not different to energy expenditure in the rest trial. Hwa et al. (1997) showed that leptin administration results in an increase in spontaneous physical activity in mice. In addition, ghrelin administration leads to suppressed spontaneous physical activity in rats (Tang-Christensen et al. 2004). Serum leptin and plasma ghrelin concentrations between 38 and 62 hours after exercise (when PAEE increased) were not measured in this study. It is possible that exercise at 40%  $\dot{V}O_2\text{max}$  might alter energy balance hormone concentrations such as leptin or ghrelin concentrations and that this could influence PAEE in sedentary men.

In this study, there was no change in energy intake (rice cakes and nutella) after exercise either at 40% or 70%  $\dot{V}O_2\text{max}$ . This is consistent with previous studies showing that there was no difference in the energy intake between moderate and high intensity exercise during a post-exercise buffet meal (King et al. 1994; Erdmann et al. 2007; Imbeault et al). In contrast, another study found that high intensity exercise induced suppression of energy intake during a buffet meal (Kissileff et al. 1999). Additionally, there was no impact of the different intensity exercise on subjective feelings of hunger and fullness in the present study. This is consistent with previous studies (Pomerleau et al. 2004; Erdmann et al. 2007; Imbeault et al. 1997). In contrast, other studies have demonstrated that subjective feelings of hunger were suppressed after high intensity

exercise (Broom et al. 2007; Broom et al. 2009; Burns et al. 2007; King et al. 1994). However, we should be somewhat cautious in the interpretation of the present findings. Energy intake immediately after exercise or rest was assessed when participants were given no choice (rice cakes and nutella) of food to consume. Participant preferences may limit the relevance of these findings and in retrospect this was a limitation. In summary, in the present study there was no impact of high and moderate intensity exercise on energy intake and subjective feelings of hunger and fullness; although there are limitations to the methods that were employed.

In the present study, there was no change in plasma acylated ghrelin concentrations immediately after moderate or high intensity exercise in sedentary men. In contrast, previous studies showed that plasma acylated ghrelin concentration and subjective feelings of hunger are suppressed immediately after high intensity exercise in well trained men (Broom et al. 2007; Broom et al. 2009). It is possible that suppression in acylated ghrelin concentration might be associated with the physical activity level of participants. In the present study, there was no change in serum leptin concentrations between rest and exercise trials at 40%  $\dot{V}O_{2max}$  and at 70%  $\dot{V}O_{2max}$  in sedentary men. These findings are consistent with previous studies which showed leptin concentrations were not affected immediately after moderate (Kyriazis et al. 2007) or high intensity exercise (Essing et al. 2000; Olive and Miller 2001). However, it was delayed and decreased at 48 hours after high intensity exercise (Essing et al. 2000; Oliver and Miller 2001). Therefore, while serum leptin concentrations did not change immediately after moderate or high intensity exercise in the sedentary men in the present study, it is possible that circulating leptin concentration might increase in the days after exercise.

In conclusion, there was a trend for physical activity energy expenditure to increase between 38 and 62 hours following 40%  $\dot{V}O_{2max}$  in sedentary young males. However, the sample size was small and there was considerable inter-individual variation. Little is known about the mechanism underlying this. It is possible moderate intensity exercise might alter circulating energy balance hormones and therefore influence PAEE; but hormones were not measured in the hours after exercise. Therefore, future research should investigate this further. Additionally, while there was no difference in

immediate post-exercise energy intake between rest and exercise trials, this was based on limited food choices and therefore this also needs further research.

## CHAPTER 4

### THE IMPACT OF EXERCISE AT 40% $\dot{V}O_{2\max}$ ON ENERGY INTAKE, PHYSICAL ACTIVITY ENERGY EXPENDITURE AND ENERGY BALANCE HORMONES IN SEDENTARY MALES AGED BETWEEN 18 AND 34 YEARS

#### 4.1 Introduction

The previous study demonstrated that there was a trend for an increase in PAEE between 38 and 62 hours after moderate intensity exercise (40%  $\dot{V}O_{2\max}$ ) in sedentary men (Chapter 3). However, little is known about the mechanisms which underlie this. Exercise may have an impact on behaviours such as energy intake and PAEE by influencing the secretion of certain hormones (McMurray and Hackney 2005). It is possible that a single bout of moderate intensity exercise might alter concentrations of energy balance hormones such as leptin and adiponectin and this could subsequently influence PAEE in sedentary men.

There are several hormones that may influence PAEE. Leptin is released from adipocytes and influences energy expenditure (Novak and Levine 2007). It has been demonstrated that administration of leptin into the cerebroventricular area of the brain can stimulate spontaneous physical activity in rats (Choi et al. 2008) and that serum leptin concentrations decrease after vigorous intensity exercise (Landt et al. 1997; Duchlos et al. 1999; Koistinen et al. 1998) and even up to 48 hours after exercise (Essing et al. 2000). Adiponectin is a hormone that is released from adipocytes. It has been shown that physical activity energy expenditure estimated using an accelerometer is related to adiponectin concentrations (St-Pierre et al. 2006) and that the concentration of adiponectin changes after maximal exercise in highly trained male rowers (Jurimae et al. 2007). Orexin A (hypocretin 1) is released from the lateral and dorsomedial hypothalamus and this hormone is expressed in many areas such as the hypothalamic area, extra thalamic brain site, and spinal cord (Taske et al. 2008). An increase in spontaneous physical activity has been reported after injection of orexin A in the paraventricular nucleus of rats (Kiwaki et al. 2004). Orexin A in cerebrospinal fluid increased after exercise in dogs (Wu et al. 2002). However, there was no difference in



the concentration of orexin A in blood and cerebrospinal fluid in rats (Lin et al. 2005). In contrast, ghrelin administration leads to decreased spontaneous physical activity in rats (Tang-Christensen et al. 2004). Therefore, it is possible that leptin, adiponectin and orexin A concentrations might influence PAEE and this might explain previous findings after moderate intensity exercise (Chapter 3). Additionally, the investigation of the impact of moderate intensity exercise on PAEE might also provide a further opportunity to investigate the relationship between exercise and energy intake.

This study aimed to investigate the impact of moderate intensity exercise (40%  $\dot{V}O_{2\max}$ ) on post-exercise PAEE, energy balance hormones and energy intake in sedentary men. The main hypothesis of this study is that PAEE will increase in the day following exercise and thus confirm the trend observed in the previous study (Chapter 3). Secondly, the increase in PAEE might be explained by changes in energy balance hormones such as leptin and adiponectin.

## 4.2 Methods

### 4.2.1 Participant recruitment and eligibility

Table 4.1 Participant characteristics.

	Mean±SE
Age (years)	28±2
Body mass (kg)	72.0±2.5
Height (cm)	181.5±1.9
BMI (kg/m <sup>2</sup> )	22±1
Waist (cm)	79.2±1.7
Hip (cm)	96.9±1.2
Waist/Hip ratio	0.8±0.0
Total percent body fat (%)	18±1
Total fat mass (kg)	13 ±1
Total lean mass (kg)	55±2
Blood pressure: systolic/ diastolic (mmHg)	120±3/78±3
Resting heart rate (beats /min)	68±3
$\dot{V}O_{2\max}$ (ml/kg/min)	49.8±1.7
Physical activity level (PAL)	1.6±0.2
Activity > 4.8 METs (minutes/day)	13±4

Values are means ± SE (n=10). BMI, body mass index;  $\dot{V}O_{2\max}$ , maximal oxygen consumption. Body composition determined by dual-energy x-ray absorptiometry (DEXA). Physical activity level (PAL) and metabolic equivalent (METs) estimated from a physical activity monitor (Actiheart) worn for seven days.

Ten sedentary males aged between 18 and 34 years participated in this study which had approval from the Local NHS Ethics Committee (Table 4.1). In order to assess eligibility, participants completed a health history questionnaire and a Physical Activity Readiness Questionnaire (PAR-Q). The participants were non-smokers with a body mass index of less than 30 kg/m<sup>2</sup>. Furthermore, their body mass had not changed by more than 5 kg in the last six months. In addition, to be eligible to take part, they had to confirm that they did not do any moderate intensity activity more than 30 minutes per day and no more than three times per week (from self report, Appendix 1). The

participants provided written informed consent. Participants wore a physical activity monitor for seven days in order to assess physical activity energy expenditure. As shown in Table 4.1, the participants were low active with a physical activity level (PAL) lower than 1.6 (Brooks et al. 2004). Additionally, these data were also analysed to determine the intensity of physical activity. No participants spent more than 30 minutes every day above 4.8 METs (Defined as moderate intensity physical activity, Pollock et al. 1998).

#### **4.2.2 Experimental design**

On one occasion participants performed a standardised bout of exercise and on another occasion they rested in the laboratory for the same period of time; each trial was separated by at least one week (Figure 4.1). In the exercise trial, participants walked on a treadmill at a target exercise intensity of 40%  $\dot{V}O_{2\max}$  for 60 minutes. This exercise model was based on the results of Chapter 3.

#### **4.2.3 Energy intake**

A buffet meal was provided one hour following rest or exercise main trials. Participants were given 30 minutes to consume food from the buffet. The buffet was designed to be similar to typical lunchtime foods (Appendix 5). Additionally, energy intake outside the laboratory was assessed. Participants weighed and recorded their daily food intake on the main trial day and for two days after each main trial (General Methods).

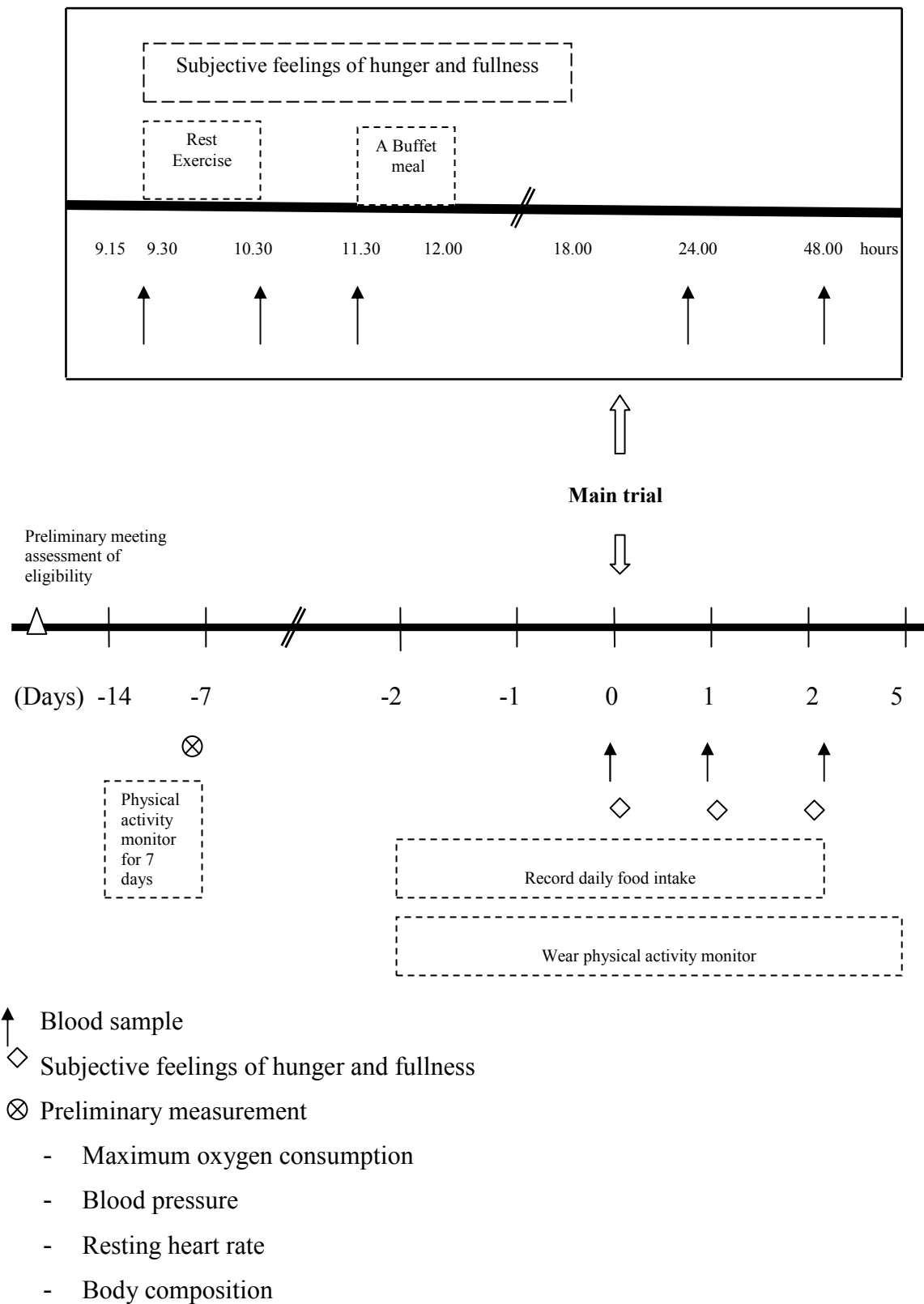


Figure 4.1 Study protocol. Main trial represents either rest or exercise (40%  $\dot{V}O_2\text{max}$ ) in a randomized order with at least one week between trials.

#### **4.2.4 Subjective feelings of hunger and fullness**

As explained in the general methods (Chapter 2) subjective feelings of hunger and fullness (Appendix 3) were assessed using 100 mm visual analogue scales (VAS). There were assessed before and after exercise as well as after eating on each main trial day. In addition, to measurements taken in the laboratory, subjective feelings of hunger and fullness were assessed between 12.00 p.m. and 6 p.m. Timers were provided in order to remind participants to take this measure every 30 minutes. This measure was repeated at 24 and 48 hours.

#### **4.2.5 Energy expenditure and physical activity energy expenditure**

Expired gas was collected for one minute every ten minutes in the exercise trial and for five minutes in the rest trial in order to estimate energy expenditure using indirect calorimetry. Further details on these measurements can be found in the general methods (Chapter 2). To examine physical activity energy expenditure outside the laboratory, participants wore a physical activity monitor (Actiheart, Cambridge Neurotechnology Ltd., Cambridge) on the main trial day and also two days following each main trial (Chapter 2). PAEE in classified metabolic equivalent (METs) zones (General Methods) was analysed in one, 10 and 30 minute epochs.

#### **4.2.6 Energy intake and physical activity energy expenditure control**

Participants were asked to replicate their food and fluid intake for two days before each main trial day. The participants were also asked to avoid alcohol and caffeine for 48 hours before each main trial. In addition, participants wore a physical activity monitor and avoided strenuous exercise for two days before each main trial. Average physical activity energy expenditure for two days prior to each trial did not differ significantly between trials (Rest,  $686 \pm 70$  kcal; Exercise,  $761 \pm 122$  kcal,  $P=0.44$ ).

#### 4.2.7 Statistical analysis

Sample size was estimated by using previous work that shows that the level of leptin 48 hours following acute exercise is 0.6 ng/ml greater than a control trial where the SD of the difference is 0.2 ng/ml (Essig et al. 2000). Therefore, to detect a mean difference of 0.6 ng/ml in the present trial required a estimated sample size of 12 and allowing for two participants to dropout (80% power and 5% alpha).

All data were analysed by using Statistical software (SPSS 14.0, United States). Two-way repeated measures ANOVA and *t*-tests were used to analyses the differences for leptin, acylated ghrelin, adiponectin, blood glucose, blood lactate concentrations and subjective feelings of hunger and fullness between trials over period of time. Paired *t*-tests were used for *post hoc* comparisons with a Bonferroni method. Statistical significance was accepted at the 5% level ( $P < 0.05$ ). Data are presented as means $\pm$ SE.

### 4.3 Results

#### 4.3.1 Trial data

	Rest	Exercise
Environmental		
- Barometric pressure (mmHg)	743±2	744±2
- Temperature (°C)	19±0	19±0
- Humidity (%)	60±2	63±3
$\dot{V}O_2$ (ml/kg/min)	3.8±0.1	19.5±0.5*
Energy expenditure (kcal)	88±6	431±40*
Energy expenditure/BW (kcal/kg)	1.2±0.1	6.0±0.5*
Total carbohydrate oxidation (g)	8.1±2.6	80.8±15.4*
Total fat oxidation (g)	6.0±0.9	11.2±3.5*
Water intake (ml)	316±58	578±82*

Table 4.2 Trial data in rest and exercise trials. Values are means±SE (n=10).  $\dot{V}O_2$ , average oxygen consumption every ten minutes; water intake, during rest or exercise trials; BW, body weight. \*, significantly different between rest and exercise at 40%  $\dot{V}O_{2max}$  ( $P<0.05$ ).

From questionnaires prior to each trial (Appendix 2), student's *t*-test revealed that sleeping and overnight fast time were not different between rest and exercise trials. There was no significant difference in the environmental temperature (Student's *t* test,  $P=0.63$ ), barometric pressure ( $P=0.62$ ) and humidity ( $P=0.31$ ) between the rest and the exercise trials. As can be seen from Table 4.2, participants expended approximately 431 kcal in the exercise trial.

### 4.3.2 Energy intake

	Rest	Exercise
Energy intake (kcal)	1481±168	1499±166
Carbohydrate (g)	237±34	227±29
Lipid (g)	49±16	56±10
Protein (g)	56±6	51±6
Relative energy intake (kcal)	1481±168	1155±170
EI/Body weight (kcal/kg)	20.2±2.0	20.8±2.2
REI/Body weight (kcal/kg)	20.2±2.0	16.1±2.2

Table 4.3 Absolute and relative energy intake at lunch time on the trial days during a buffet meal. Values are means±SE (n=10). Relative energy intake (REI) = energy intake-(total energy cost of the exercise session-(exercise time x resting energy expenditure)). EI, energy intake.

After one hour of rest or exercise, a buffet meal was provided for a period of 30 minutes. Even though relative energy intake after exercise was considerably less than the rest trial (22%), this was not statistically significant ( $P=0.09$ ). As shown in Table 4.3, there was no significant difference in an absolute energy intake between rest and exercise trials ( $P=0.91$ ).



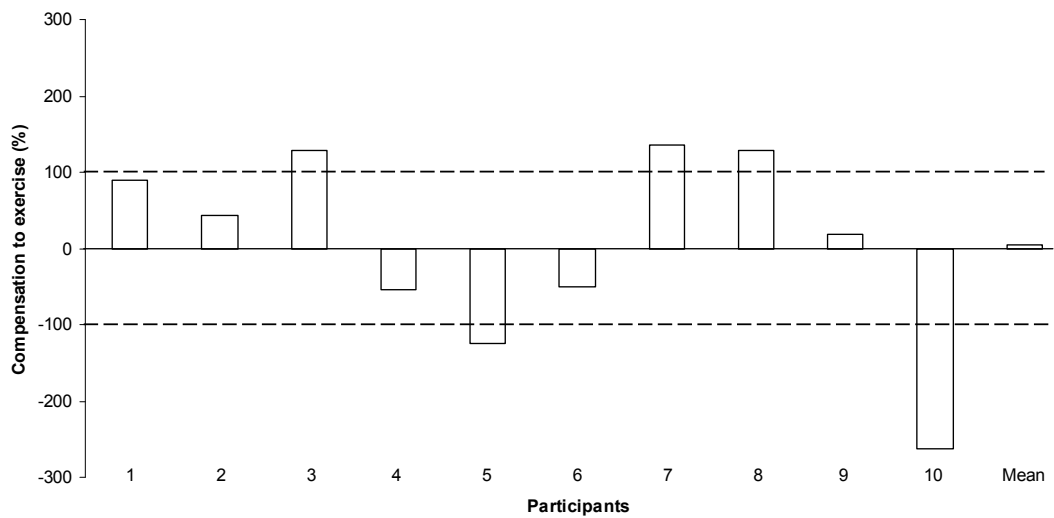


Figure 4.2 Compensation of exercise-induced energy expenditure during a buffet meal. Compensation= [(energy intake during the buffet meal in each exercise trial - energy intake in control rest trial)/exercise cost of exercise above resting energy expenditure] x 100. A value of 0% represents energy intake after exercise being equivalent to energy intake after the rest trial; a value of 100% represents energy intake after exercise being 100% greater than after rest and this is equivalent to the energy expended in the exercise session above rest; a value of -100% represents energy intake after rest being 100% greater than after exercise and is equivalent to the energy expended in the exercise session above rest (see general methods for more details).

From Figure 4.2, it can be seen that after exercise four participants showed evidence of compensation to exercise. For example, the energy intake during the exercise trial in subject one was higher than the energy intake in the rest trial and was 89% of energy expended during exercise above resting energy expenditure.

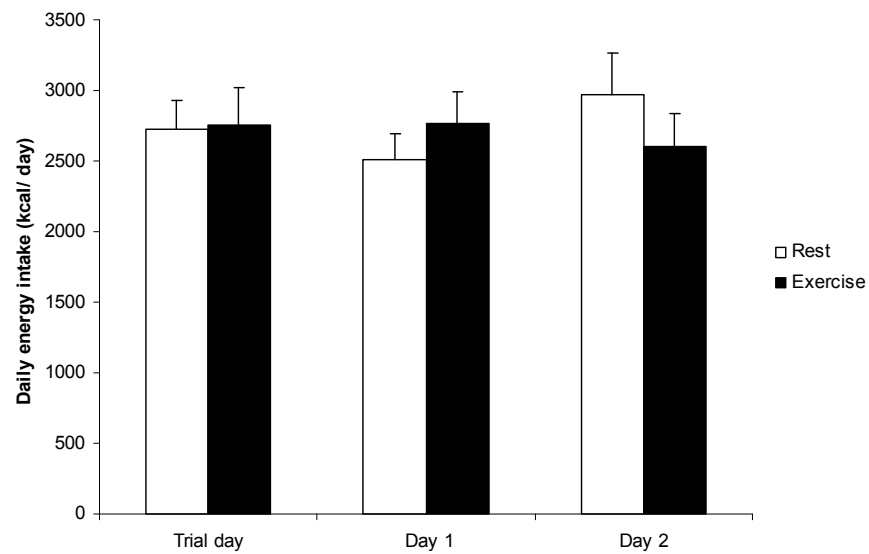


Figure 4.3 Energy intake (EI) in rest and exercise trials. EI from a buffet meal and weighed food and fluid records on each trial day; Day 1, one day after each main trial; Day 2, two days after each main trial. Values are means $\pm$ SE (n=10).

Figure 4.3 shows energy intake estimated from weighed food records. Energy intake did not differ significantly between rest and exercise trials.

### 4.3.3 Subjective feelings of hunger and fullness

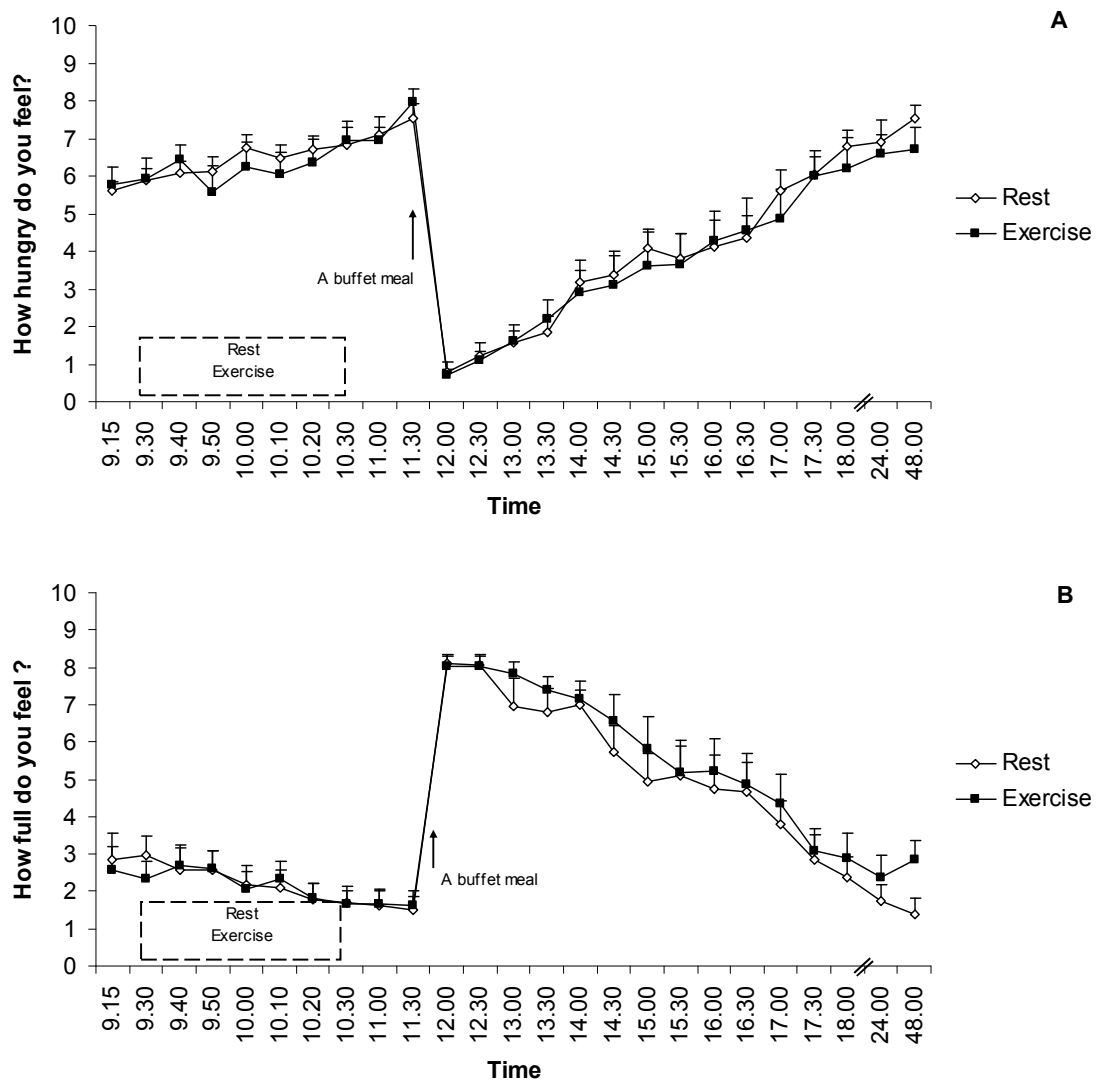


Figure 4.4 Subjective feelings of hunger and fullness using a 100 mm visual analogue scale (means $\pm$ SE) during exercise and rest trials (n=10). A, how hungry do you feel; B, how full do you feel. Data have been converted to a 0-10 scale for presentation.

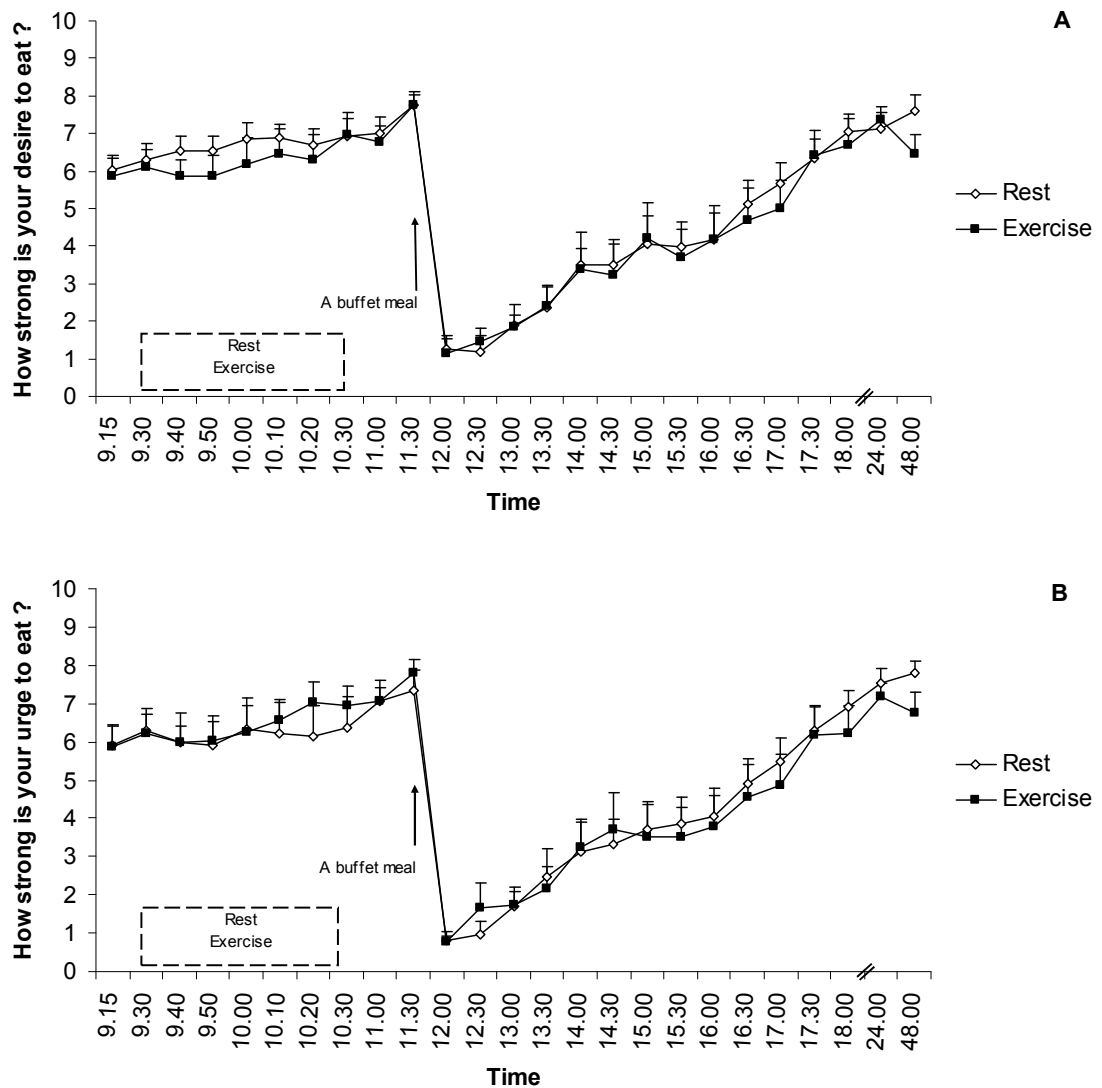


Figure 4.5 Subjective feelings of desire and urge to eat using a 100 mm visual analogue scale (means $\pm$ SE) during exercise and rest trials ( $n=10$ ). A, how strong is your desire to eat; B, how strong is your urge to eat. Data have been converted to a 0-10 scale for presentation.

Appetite was assessed both before and after exercise. Figures 4.4 and 4.5 show subjective feelings of hunger between exercise and rest trials. According to these figures, it is clear that there was no difference in subjective feelings of hunger and fullness between rest and exercise trials. There was a time effect for hunger, fullness, desire and urge to eat, as a result of consumption of the buffet meal ( $P<0.01$ ). For clarity of presentation, these have not been depicted in the figures.

#### 4.3.4 Physical activity energy expenditure

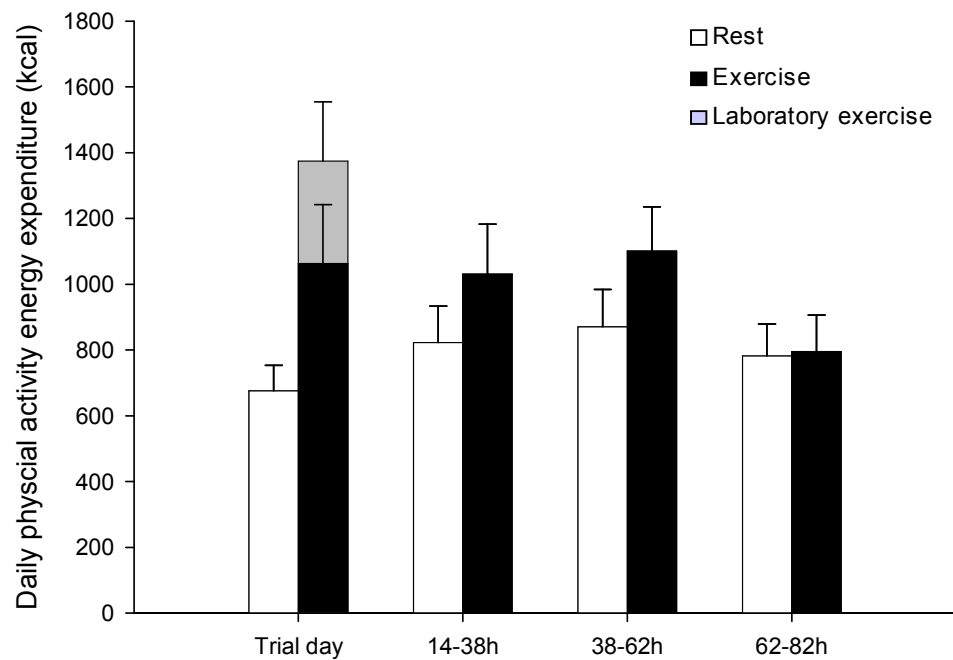


Figure 4.6 Physical activity energy expenditure (PAEE) in rest and exercise trials. Trial day, rest or exercise; 14-38h, between 14-38 hours; 38-62h, between 38-62 hours; 62-86h, between 62-86 after main trials. Values are means $\pm$ SE (n=10) except between 62-86h (n=9).

Daily physical activity energy expenditure (PAEE) is the energy expended above rest. Two-way ANOVA revealed a trend for the interaction between trial and time for PAEE after subtraction of exercise in the laboratory ( $P=0.07$ ). There was an effect of trial for PAEE ( $P=0.04$ ). Further analysis of these data showed that physical activity energy expenditure between 38 and 62 hours after exercise at 40%  $\dot{V}O_{2\max}$  was greater than rest ( $P=0.05$ ).

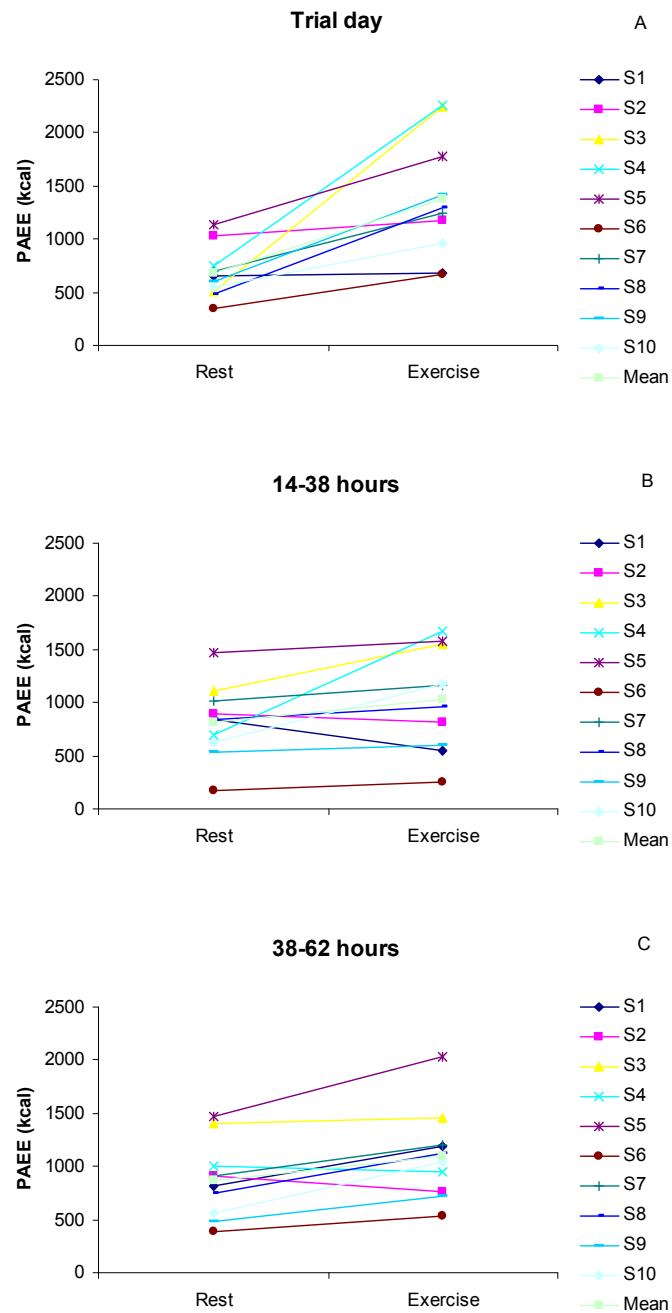


Figure 4.7 Physical activity energy expenditure (PAEE) in rest and exercise trials on each main trial day (A), 14-38h (B) and 38-62h (C). Values are means $\pm$ SE (n=10).

Figure 4.7 shows the physical activity energy expenditure between rest and exercise trials for three days. It can be seen that eight out of ten participants expended more energy through activity on the trial day, 14-38h and 38-62h after exercise at 40%  $\dot{V}O_2$ max.

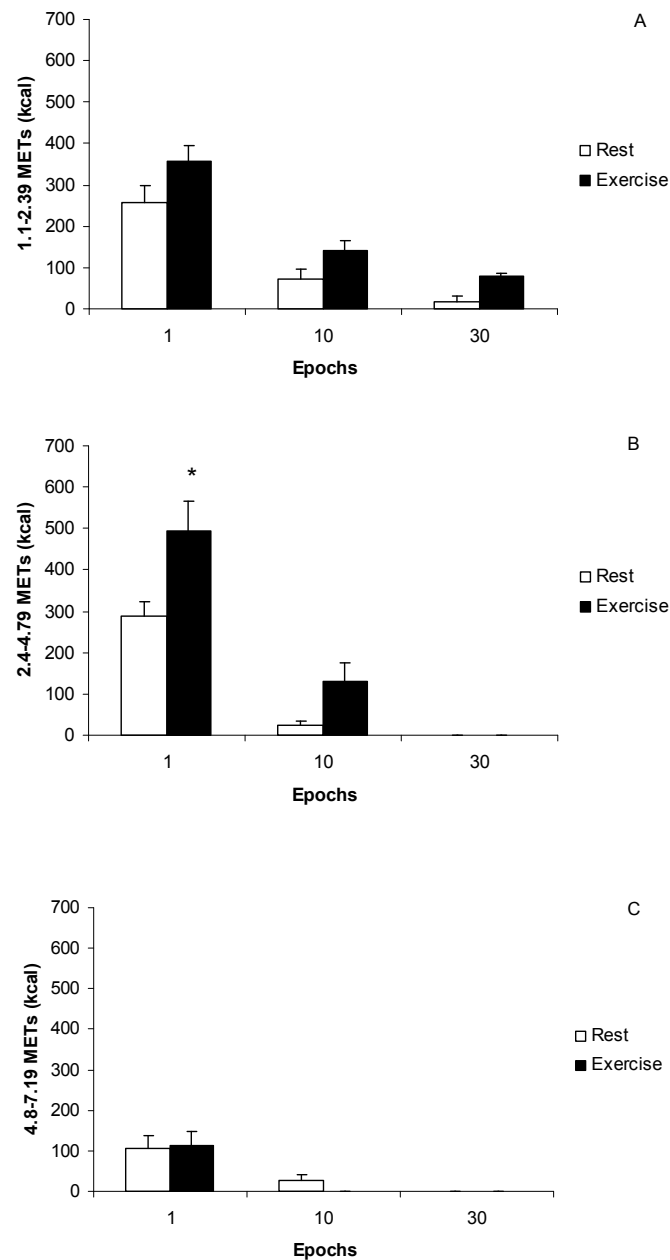


Figure 4.8 Physical activity energy expenditure on the main trial day after excluding energy expend during laboratory exercise in very light (1.1-2.39 METs, A), light (2.4-4.79 METs, B) and moderate (4.8-7.19 METs, C) intensity activity. Values are means $\pm$ SE (n=10). \*, significantly different between rest and exercise trials at 40%  $\dot{V}O_2$ max ( $P<0.05$ ).

Figure 4.8 presents PAEE on the main trial day. As can be seen from the graph, energy expended during one minute epochs in light intensity activity increased by 71% ( $P=0.02$ ) following exercise. However, there was no change in energy expended in hard (7.2-10.19 METs) and very hard ( $\geq 10.2$  METs) intensity activity between rest and exercise trials (data not shown).

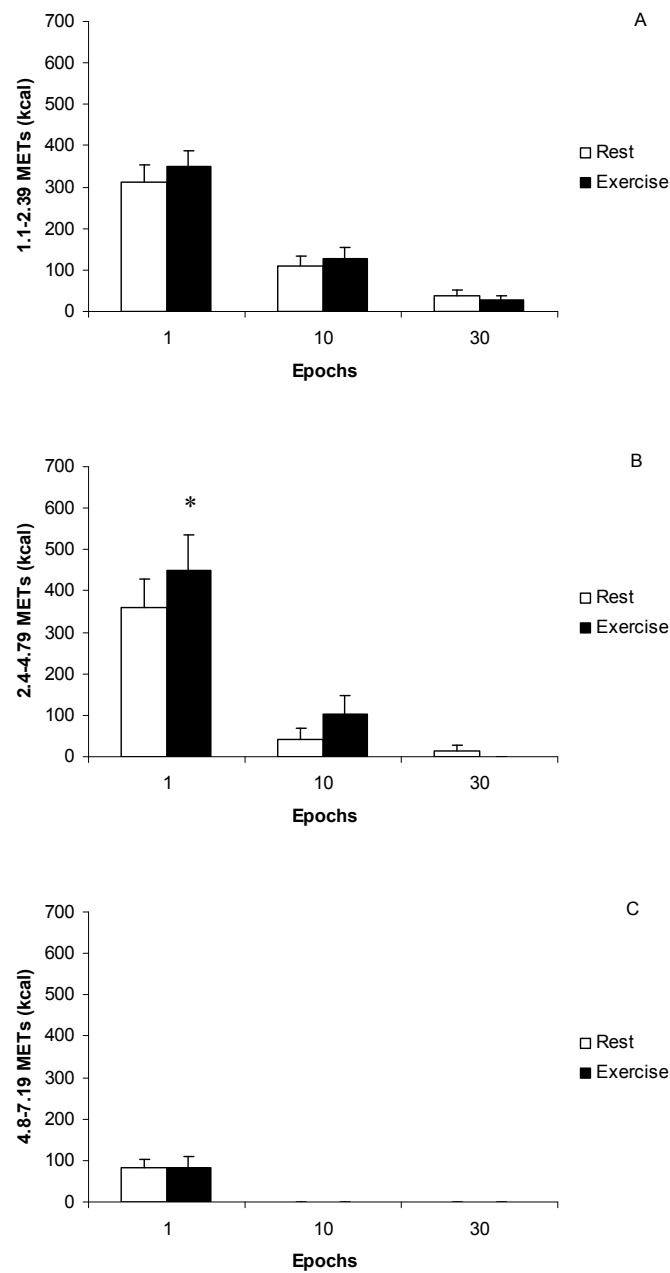


Figure 4.9 Physical activity energy expenditure between 14 and 38 hours after exercise and rest trials in very light (1.1-2.39 METs, A), light (2.4-4.79 METs, B) and moderate (4.8-7.19 METs, C) intensity activity. Values are means $\pm$ SE (n=10). \*, significantly different between rest and exercise at 40%  $\dot{V}O_2$ max ( $P<0.05$ ).

Figure 4.9 presents PAEE at three different intensities between 14 and 38 hours after exercise or resting. It can be seen from Figure 4.9 that there was a 24% increase in energy expended for one minute epochs in light intensity activity ( $P=0.01$ ). PAEE in very light (1.1-2.39 METs), moderate (4.8-7.19 METs), hard (7.2-10.19 METs) and very hard ( $\geq 10.2$  METs) intensity activity did not differ significantly between rest and exercise trials (data not shown for hard and very hard).



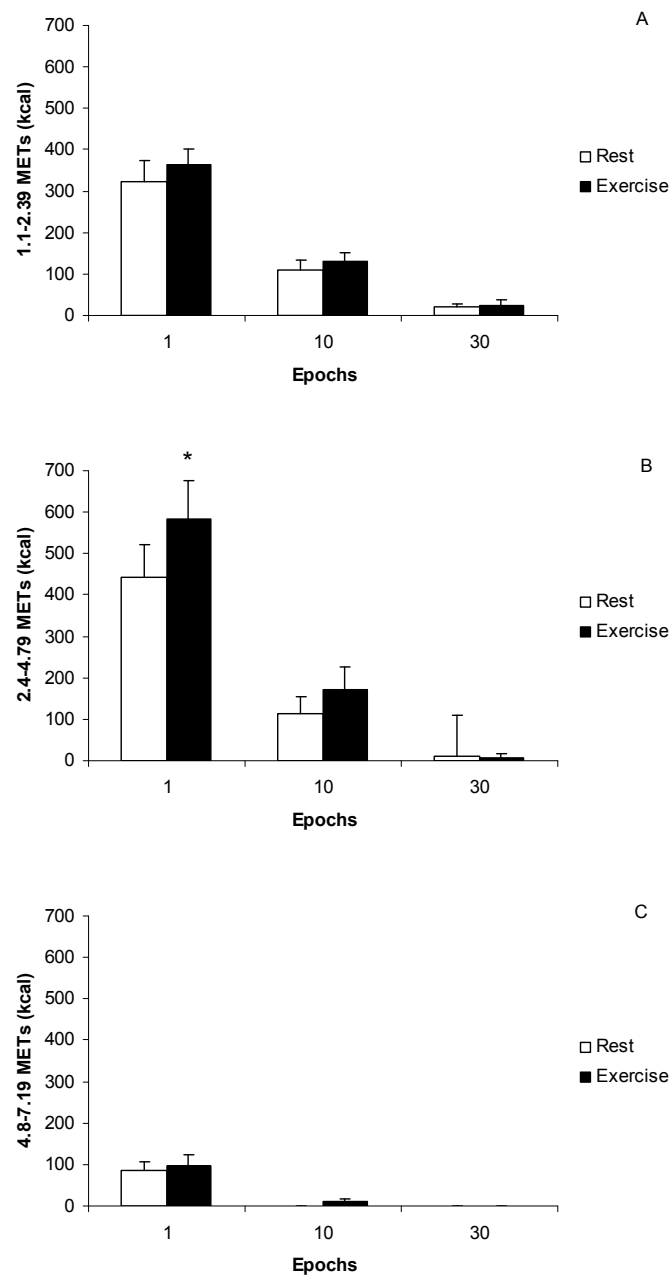


Figure 4.10 Physical activity energy expenditure between 38 and 62 hours after exercise and rest trials in very light (1.1-2.39 METs, A), light (2.4-4.79 METs, B) and moderate (4.8-7.19 METs, C) intensity activity. Values are means $\pm$ SE (n=10). \*, significantly different between rest and exercise at 40%  $\dot{V}O_2$ max ( $P<0.05$ ).

As shown in Figure 4.10, there was a 31% increase in energy expended ( $P=0.03$ ) for one minute epochs in light intensity activity between 38-62 hours after the exercise trial. However, energy expended at very light (1.1-2.39 METs), moderate (4.8-7.19 METs), hard (7.2-10.19 METs) and very hard ( $\geq 10.2$  METs) intensity activity was not statistically different between two trials (data not shown for hard and very hard).

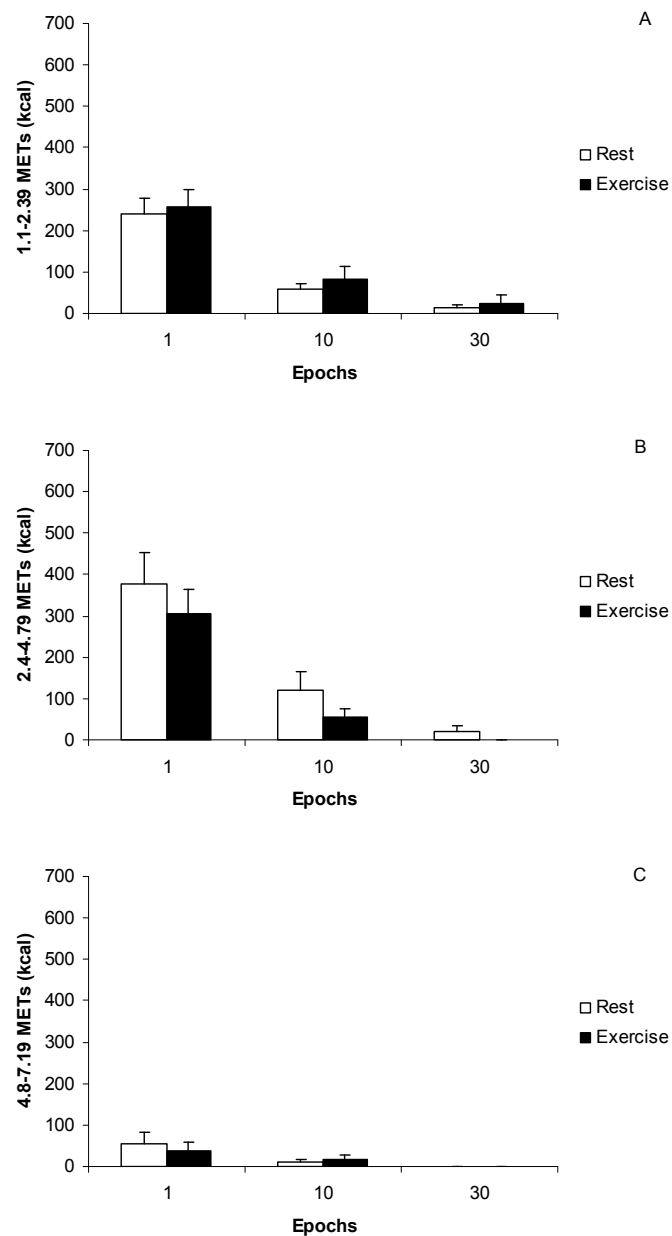


Figure 4.11 Physical activity energy expenditure between 62 and 86 hours after exercise and rest trials in very light (1.1-2.39 METs, A), light (2.4-4.79 METs, B) and moderate (4.8-7.19 METs, C) intensity activity. Values are means $\pm$ SE (n=9).

Figure 4.11 shows physical activity energy expenditure between 62 and 86 hours after rest or exercise trials in different MET zones. It can be seen that there was no difference in energy expended in very light, light and moderate intensity activity between two trials at this time point.

## 4.3.5 Blood parameters

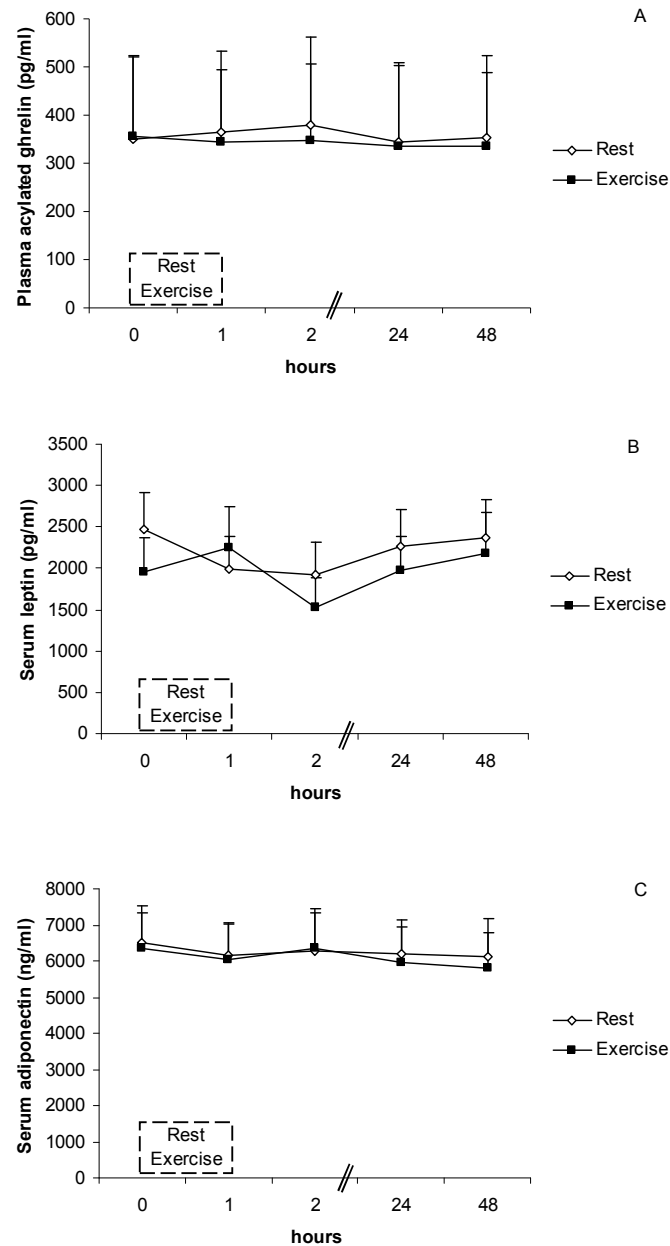


Figure 4.12 Plasma acylated ghrelin (A), serum leptin (B) and serum adiponectin concentrations (C) during rest and exercise trials. Value are means $\pm$ SE (n=10).

Two-way ANOVA indicated a trial  $\times$  time interaction ( $P=0.05$ ) for serum leptin concentrations. *Post hoc* test showed a difference between trials one hour after exercise for leptin concentration ( $P=0.05$ ), but not after adjustment using the Bonferroni method. As shown in Figure 4.12, it can be seen that there was no change in plasma acylated ghrelin and serum adiponectin concentrations immediately, one, 24, and 48 hours after rest or exercise trials.

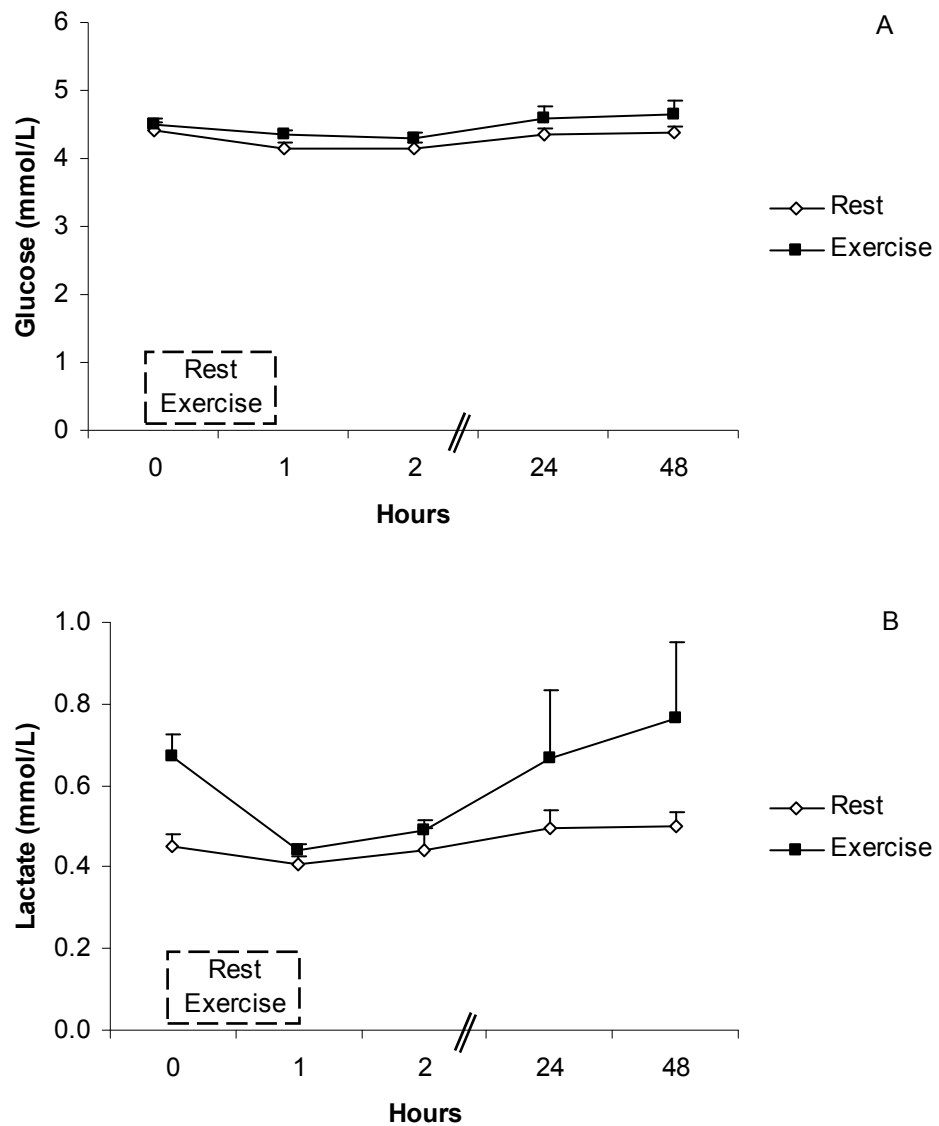


Figure 4.13 Blood glucose (A) and lactate (B) concentrations in rest and exercise trials. Value are means $\pm$ SE (n=10).

Figure 4.13 presents blood glucose and lactate concentration in exercise and resting trial. There was no significant difference in plasma blood glucose and blood lactate concentrations between trials.

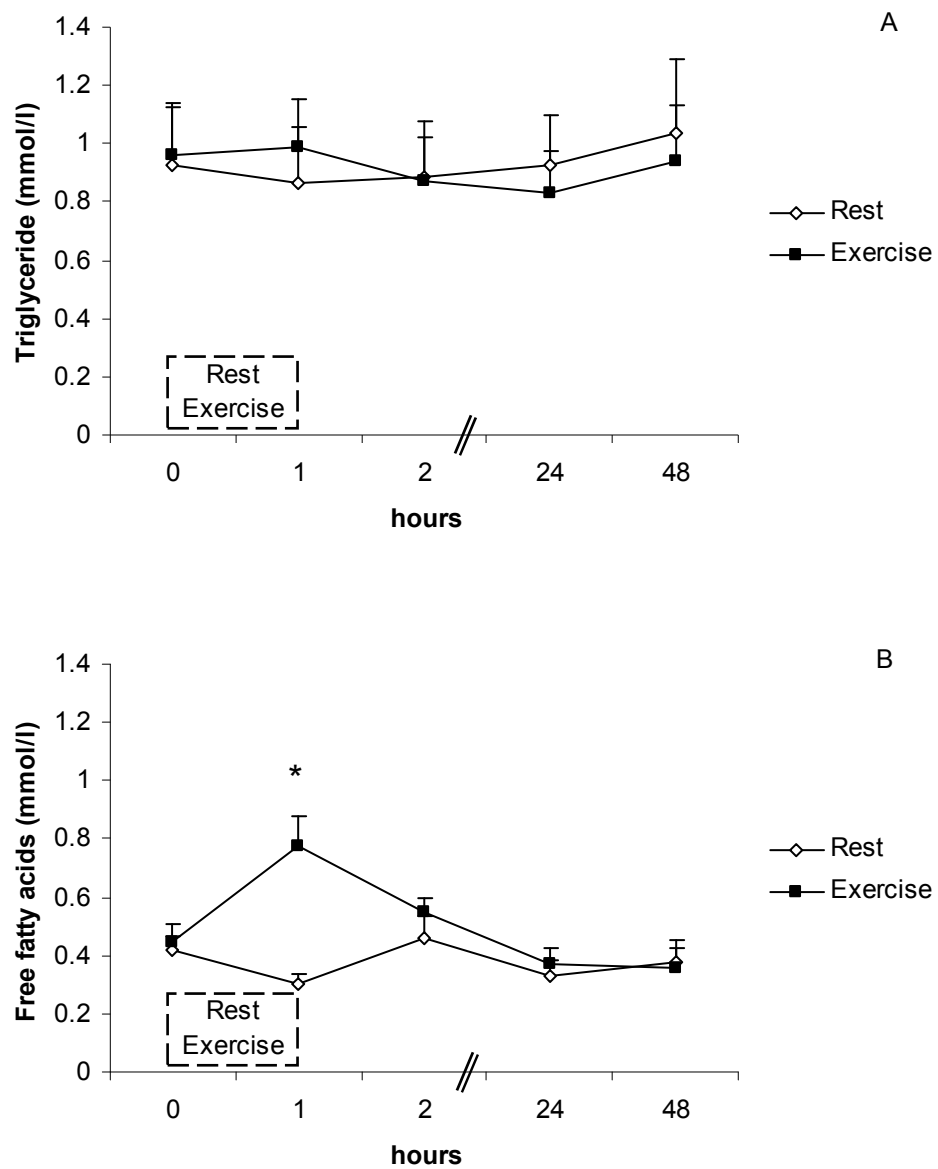


Figure 4.14 Triglyceride (TAG, A) and free fatty acids (FFA, B) concentrations in rest and exercise trials. Value are means $\pm$ SE (n=10). \*, significantly different between rest and exercise at 40%  $\dot{V}O_2\text{max}$  ( $P < 0.05$ ).

Two-way ANOVA showed a trial x time interaction ( $P < 0.01$ ) for plasma free fatty acids concentrations. Plasma free fatty acids concentration rose significantly by 72 % ( $P = 0.01$ ) immediately after exercise and fell slowly in the recovery period. However, there was no significant difference in plasma triglyceride concentration between trials.

#### 4.4 Discussion

This study aimed to investigate the impact of a single bout of moderate intensity exercise ( $40\% \dot{V}O_{2\max}$ ) on physical activity energy expenditure (PAEE), energy balance hormones and energy intake in young sedentary males. Interestingly, PAEE was increased until 62 hours following moderate intensity exercise in sedentary males. However, there was no difference in any circulating energy balance hormones such as acylated ghrelin, leptin and adiponectin. Additionally, there was no change in energy intake during a buffet meal following a single bout of moderate intensity exercise or energy intake for two days following exercise.

A main finding of the present study is that daily free-living PAEE increased following exercise due to increased energy expended in light intensity activity (2.4-4.79 METs) such as standing and walking. The increase in PAEE in light intensity activity was relatively short-lived and had disappeared 62h after exercise. This is consistent with the findings of the previous chapter, which showed that there was a trend for daily PAEE to increase between 38 and 62 h after moderate intensity exercise in sedentary men. One study reported that energy expenditure between 1 and 7 p.m. assessed using a heart rate monitor the day after moderate intensity exercise was higher than after high intensity exercise but there was no difference when compared with rest (Kriemler et al. 1999). To our knowledge, this is the first study to show that PAEE in light intensity activity increases over two days following a single moderate intensity bout of exercise in sedentary young males.

Hagobian et al. (2008) demonstrated that daily PAEE did not change during four bouts moderate intensity exercise in obese males and females. Other studies found that there was no change in non-exercise PAEE during exercise training in sedentary (Hollowell et al. 2009) and obese participants (Blaak et al. 1992). It is possible that the increase in PAEE in this study might be an acute phenomenon to the first novel bout of exercise in sedentary individuals and that such an effect disappears over time as exercise becomes more familiar.

In this study, there was no impact of a single bout of exercise on absolute energy intake during a buffet meal. This finding was consistent with previous studies showing no short term compensation in energy intake and macronutrient intake following low and moderate intensity exercise during a buffet meal (Pomerleau et al. 2004; Imbeault et al. 1997; King et al. 1994). In contrast, one study demonstrated that energy intake during a buffet meal after walking for 60 minutes was lower than after rest (George and Morganstein 2003). Additionally, in the present study energy intake was not altered two days following moderate intensity exercise. This is consistent with a previous study (Jamurtas et al. 2004). Subjective feelings of hunger did not change in the present study. This is consistent with previous studies which reported that there was no change in subjective feelings of hunger and fullness after low intensity and high intensity exercise (Erdmann et al. 2007; Imbeault et al. 1997). However, other studies have shown subjective feelings of hunger to be suppressed immediately after high intensity exercise (Broom et al. 2007; Broom et al. 2009; Burns et al. 2007; King et al. 1994). Therefore, the effect of a single bout of exercise on energy intake and subjective feelings of hunger and fullness remains unclear. In the present study, it appears that an acute bout of exercise at 40%  $\dot{V}O_{2max}$  did not induce changes in energy intake or subjective feelings of hunger and fullness.

There are several energy hormones which might affect PAEE (e.g., leptin, adiponectin and orexin A concentrations). Leptin and adiponectin concentrations did not change in this study, which is similar to the findings of other studies (Ractte et al. 1997; Kyriazis et al. 2007; Hicky et al. 1996; Essing et al. 2000; Ferguson et al. 2004; Kraemer et al. 2004). We attempted to measure plasma orexin A but concentrations were mostly not detectable. It is possible that the intensity of the exercise in this study may be insufficient to induce changes in circulating leptin and adiponectin. However, this study suggests that the increase in PAEE is not associated with circulating hormones such as leptin and adiponectin.

There was no change in post-exercise circulating plasma acylated ghrelin concentrations after exercise and before eating in this study. Suppression of acylated ghrelin concentrations due to high intensity exercise has been demonstrated in previous studies (Broom et al. 2007; Broom et al. 2009). It appears that plasma acylated ghrelin

concentrations change after high but not moderate intensity exercise. However, an earlier study in this thesis showed no change in circulating acylated ghrelin concentration after participants expended 430 kcal during 40 minutes of high intensity exercise in sedentary men (Chapter 3). In contrast, previous studies demonstrated that circulating acylated ghrelin concentration was suppressed after expending approximately 466 kcal during 30 minutes of high intensity exercise (Broom et al. 2007). Participants in this study (Chapter 3) were sedentary and had a maximum oxygen consumption lower than reported for participants in previous studies (Broom et al. 2007; Broom et al. 2009). Active men might have greater suppression of acylated ghrelin in response to high intensity exercise than sedentary men. In summary, there was no impact of a single bout of moderate intensity exercise on energy intake and there was no change in plasma acylated ghrelin concentrations and blood glucose concentration.

Behavioural, physiological and psychological processes are regulated by the brain (Dishman et al. 2006). However, little is known about the mechanisms underlying the impact of exercise on the function of the central and peripheral neural systems (Dishman et al. 2006). It is possible that exercise might alter behaviour by changing chemicals in the brain. PAEE is influenced by not only physiological but also psychological factors (Bryan et al. 2007; Berthoud 2007). Exercise changes the subjective experience such as mood and perceived exertion and therefore motivation to exercise by changing attitude toward exercise, intentions to exercise and self efficacy (Bryan et al. 2007). It has been suggested that anxiety and depression are reduced after a single session of exercise and that the changes in anxiety, depression and mood states after exercise are explained by changing endorphin concentrations (Guszkowska et al. 2004). Therefore, the increase in PAEE reported in the present study may reflect these changes. However, it is still unclear whether the increase in PAEE following exercise is influenced by exercise per se or perhaps secondary to improvements in self efficacy (King et al. 2007). It is possible that an acute bout of exercise may improve self efficacy and therefore induce participants to do more activity after exercise. However, the increase in light intensity PAEE in this study was of a short duration (less than ten minutes). Therefore, this might reflect spontaneous rather than conscious exercise.



There were inter-individual differences in the response to acute exercise and this needs future investigation. For instance, some participants ate less after exercise whilst other participants ate more after exercise during a buffet meal. Most participants had poor compensation of energy intake after a single bout of moderate intensity exercise. This was similar to a previous study where there were individual differences in compensation to acute exercise (Blundell et al. 2003). The effect of exercise on weight loss is associated with individual responses to exercise (Stubbs et al. 2004) and this might be one reason why some people fail to lose weight with exercise (King et al. 2007). It appears that an acute exercise-induced energy deficit and energy intake are uncoupled in young sedentary males in this study. Long et al. (2002) have shown that habitual exercisers improve the sensitivity of short-term regulation of energy intake. Therefore, the compensation of energy intake following exercise might be associated with physical activity level.

There are some limitations to this study. We are unable to conclude why sedentary males became more active after a single bout of moderate intensity exercise. This might be because we cannot measure hormones in the brain which might explain the present findings. There are also several other factors that influence PAEE and energy intake such as sleeping time, overnight fasting time, occupation, season as well as time and day of the week. However, there was no occupational difference between participants in this study and there was no difference in sleeping time, overnight fasting time, season, time, temperature and day of the week between rest and exercise trials.

In conclusion, PAEE increased over two days following exercise due to an increase in light intensity activity (e.g., standing and walking). The increase in PAEE could not be explained by circulating energy balance hormones such as leptin and adiponectin concentrations. Additionally, there was no impact of a single bout of moderate intensity exercise on absolute energy intake either during a buffet meal or over the following two days. Future research is required to examine the impact of an acute bout exercise in different groups of people such as active, obese and female participants.

## CHAPTER 5

### THE IMPACT OF ACUTE EXERCISE AT 40% AND 70% $\dot{V}O_{2\max}$ ON ENERGY INTAKE, PHYSICAL ACTIVITY ENERGY EXPENDITURE AND ENERGY BALANCE HORMONES IN YOUNG ACTIVE MALES

#### 5.1 Introduction

In previous research (Chapter 4), we have shown that a single-bout of exercise at 40% maximal oxygen consumption ( $\dot{V}O_{2\max}$ ) appears to alter physical activity energy expenditure (PAEE) over the following 62 hours. However, there was no effect on subsequent food intake and energy balance hormones such as acylated ghrelin, leptin and adiponectin. Importantly, the participants in this investigation were sedentary.

There is some evidence that regular exercisers have increased short-term accuracy in regulating food intake to compensate to previous energy intake (Long et al. 2002). It has been demonstrated that active people have high sensitivity to leptin than sedentary people (Cintra et al. 2007). It has been suggested that the coupling between energy intake and energy expenditure is associated with physical activity level (Martin et al. 2008). Physical activity might improve the sensitivity of the physiological satiety signal (Blundell et al. 2003). Exercise might impact on short term appetite control (Martins et al. 2007). For this reason, we hypothesized that active males might respond to a single bout of exercise differently to the sedentary males investigated in Chapter 4.

Based on this evidence, our primary hypothesis is that participants who take part in regular structured exercise might show effective regulation of subsequent energy intake and activity energy expenditure when the exercise challenge is familiar. It is possible that there is less effective regulation when regular exercisers perform exercise that is not familiar. This could also explain the relatively poor regulation seen in sedentary males in Chapter 4. To investigate this further, in the present study, we aim to investigate whether active males show good regulation of food intake and energy expenditure after an exercise challenge that is similar to their habitual physical activity. To examine whether familiarity is important, we also included an energy-matched exercise

challenge that was unfamiliar (moderate intensity exercise at 40%  $\dot{V}O_{2\max}$ ). This research will help us to understand whether regular exercise is associated with good regulation of energy balance and also whether the relative familiarity to exercise is an important factor.

## 5.2 Methods

### 5.2.1 Participant recruitment and eligibility

	Mean±SE
Age (years)	25±2
Body mass (kg)	80±2
Height (cm)	181±0
BMI (kg/m <sup>2</sup> )	24±0
Waist/Hip ratio	0.8±0
Total percent body fat (%)	17±2
Total fat mass (kg)	14±2
Total lean mass (kg)	63±3
Blood pressure: systolic /diastolic (mmHg)	129±4/76±3
Resting heart rate (beats /min )	59±2
$\dot{V}O_{2\max}$ (ml/kg/min )	61±2
Physical activity level (PAL)	2.3±0.1
Activity > 4.8 METs (minutes/day)	72±8

Table 5.1 Participant characteristics. Values are mean±SE (n=10). BMI, body mass index;  $\dot{V}O_{2\max}$ , maximal oxygen consumption. Body composition determined by dual-energy x-ray absorptionmetry (DEXA). Physical activity level (PAL) and metabolic equivalent (METs) estimated from physical activity monitor (Actiheart) worn for seven days.

Ten active males aged between 18 and 34 years (Table 5.1) were recruited by advertisement and participated in this study which had approval from the Local NHS Ethics Committee. In order to assess eligibility, participants completed a health history questionnaire and a Physical Activity Readiness Questionnaire (PAR-Q). The participants were non-smokers with a body mass index of less than 30 kg/m<sup>2</sup>. Furthermore, their body weight must not have changed by more than 5 kg in the last six months. In order to take part, volunteers had to be regularly active and to be eligible to take part, they had to confirm that they ran (60 minutes) at least once a week (from self report, Appendix 1). Participants provided informed consent. Participants wore a physical activity monitor for seven days in order to assess physical activity energy

expenditure (General Methods). As shown in Table 5.1, the participants were very active with a physical activity level (PAL) greater than 1.9 (Brooks et al. 2004). Actiheart data were analysed in order to confirm that they did at least 60 minutes of physical activity above 7.2 METs; defined as hard physical activity (Pollock et al. 1998). Additionally, these data were also analysed to determine the amount of physical activity (Activity >4.8 METs, moderate intensity physical activity).

### **5.2.2 Experimental design**

On two occasions participants performed a standardised bout of exercise and on another occasion they rested in the laboratory, each trial was separated by at least one week (Figure 5.1). In the moderate intensity exercise trial, participants walked on a treadmill at a target exercise intensity of 40%  $\dot{V}O_{2\max}$  until they had expended approximately 12 kcal/ kg body mass (~90 minutes). In the high intensity exercise trial, participants expended the same amount of total energy but at an intensity of 70%  $\dot{V}O_{2\max}$  (~60 minutes).

### **5.2.3 Energy intake**

A buffet meal was provided one hour following rest or exercise main trials. Subjects were given 30 minutes to consume food from the buffet. The buffet was designed to be similar to typical lunchtime foods and was the same as used in Chapter 4 (Appendix 5). Additionally, energy intake outside the laboratory was assessed. To do this, participants weighed and recorded their daily food intake on the main trial day and for two days after each main trial. Dietary composition was analysed using computer software (COMP-EAT version 5, Carlson Bengston Consultants Limited, London).

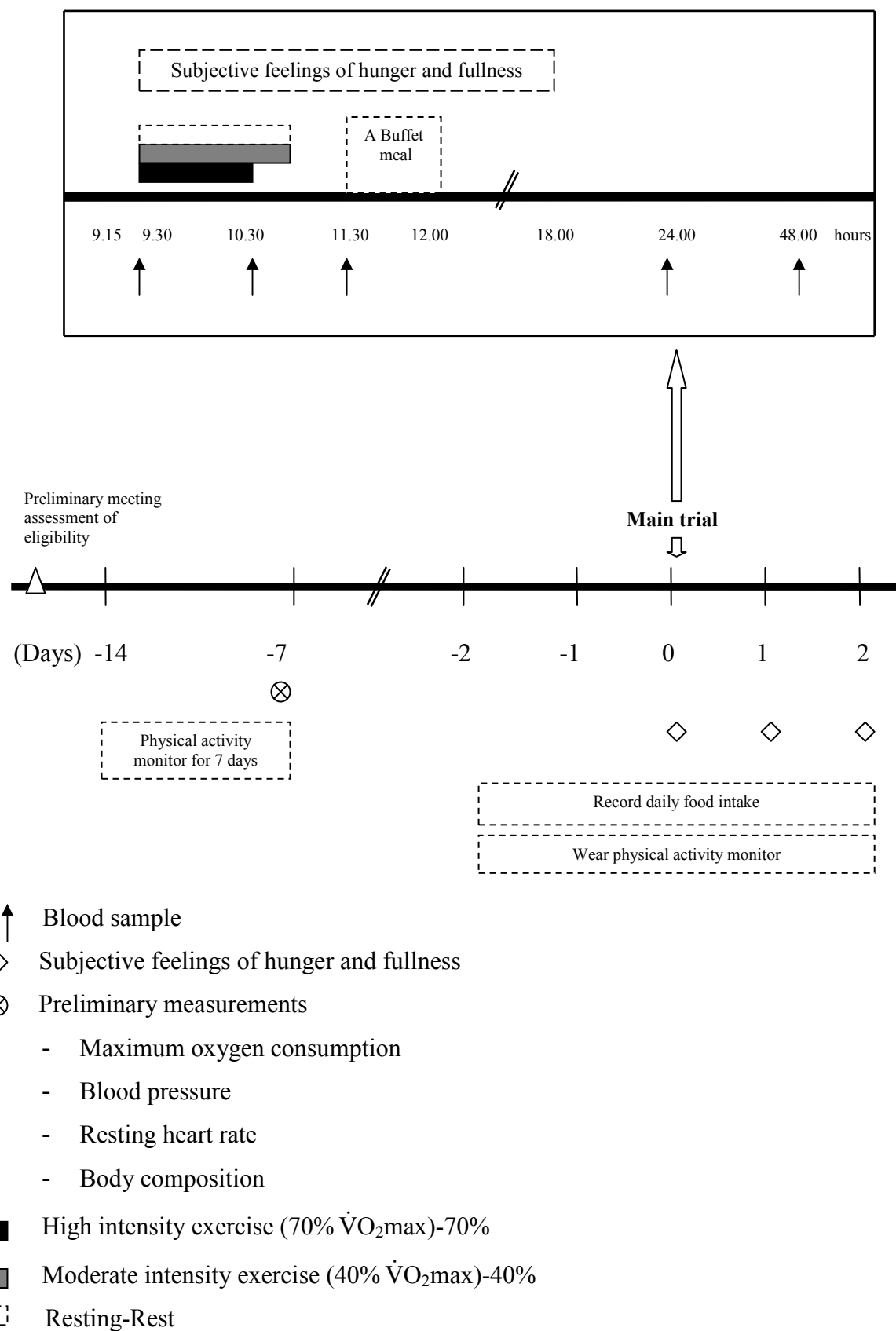


Figure 5.1 Study protocol. Main trial represents either rest 40%  $\dot{V}O_{2max}$  or 70%  $\dot{V}O_{2max}$  in a randomized order with at least one week between trials.

#### **5.2.4 Subjective feelings of hunger and fullness**

As explained in the general methods (Chapter 2), subjective feelings of hunger and fullness (Appendix 3) were assessed using 100 mm visual analogue scales (VAS). There were assessed before and after exercise as well as after eating on each main trial day. In addition, to measurements taken in the laboratory, subjective feelings of hunger and fullness were assessed between 12.00 p.m. and 6 p.m. Timers were provided in order to remind participants to take this measure every 30 minutes. This measure was repeated at 24 and 48 hours.

#### **5.2.5 Energy expenditure and physical activity energy expenditure**

During each rest or exercise main trial, expired gas was collected for one minute every ten minutes in each exercise trial and for five minutes in the rest trial in order to estimate energy expenditure using indirect calorimetry. Further details on these measurements can be found in the general methods (Chapter 2). To examine energy expenditure outside the laboratory, participants wore a physical activity monitor (Actiheart, Cambridge Neurotechnology Ltd., Cambridge) on the main trial day and also two days following each main trial (General Methods, Chapter 2). Physical activity energy expenditure (PAEE) is the energy expended above rest after subtraction of the energy spent during exercise in the laboratory.

#### **5.2.6 Energy intake and physical activity energy expenditure controls**

Participants were asked to replicate their food and fluid intake for two days before each main trial day. The participants were also asked to avoid alcohol and caffeine for 48 hours before each main trial. In addition, participants wore a physical activity monitor and avoided strenuous exercise for two days before each main trial. Average physical activity energy expenditure for two days prior to each trial did not differ significantly between trials (Rest,  $1294 \pm 186$  kcal; 40%  $\dot{V}O_{2\max}$ ,  $1550 \pm 196$  kcal; 70%  $\dot{V}O_{2\max}$ ,  $1462 \pm 220$ , all  $P > 0.24$ ).

### 5.2.7 Statistical analysis

Sample size was estimated using previous work that shows that energy intake at the first meal following acute exercise is 130 kcal greater than a control trial where the standard deviation of the difference is 170 kcal (Pomerleau et al. 2004). Therefore, to detect a mean difference of 130 kcal in the present trial requires a estimated sample size of 14 and allowing for two participants dropout (80% power and 5% alpha).

All data were analysed by using Statistical software (SPSS 14.0, United States). Student's *t*-test was used to assess the difference between trials of fasting hormones concentrations, response to exercise and energy intake. Two-way repeated measures ANOVA and *t*-tests were used to analyses the differences of total PYY, leptin, acylated ghrelin, blood glucose, blood lactate concentrations and subjective feelings of hunger and fullness between trials. Paired *t*- tests were used for *post hoc* comparisons with a Bonferroni method. Statistical significance was accepted at the 5% level ( $P < 0.05$ ). Data are presented as means $\pm$ SE.



### 5.3 Results

#### 5.3.1 Trial data

	Rest	40%	70%
Environmental			
- Barometric pressure (mmHg)	739±4	739±3	741±1
- Temperature (°C)	19±0	19±0	19±0
- Humidity (%)	43±3	40±3	45±4
$\dot{V}O_2$ (ml/kg/min)	3.8±0.2	26.6±1.3 <sup>a</sup>	40.5±1.6 <sup>b,c</sup>
Trial Time (min)	90±0	89±3	59±1 <sup>b,c</sup>
Energy expenditure (kcal)	144±18	902±41 <sup>a</sup>	903±36 <sup>b</sup>
Total carbohydrate oxidation (g)	15±7	122±18 <sup>a</sup>	143±12 <sup>b</sup>
Total fat oxidation (g)	9±1	44±6 <sup>a</sup>	35±4 <sup>b</sup>

Table 5.2 Trial data in rest and exercise trials (n=10). Values are means±SE.  $\dot{V}O_2$ , average oxygen consumption every ten minutes. Trial time; Rest, sitting for 90 minutes; Exercise, duration of exercise. 40%, exercise at 40%  $\dot{V}O_{2max}$ ; 70%, exercise at 70%  $\dot{V}O_{2max}$ . <sup>a</sup>, significantly different between rest and exercise at 40%  $\dot{V}O_{2max}$ ; <sup>b</sup>, significantly different between rest and exercise at 70%  $\dot{V}O_{2max}$ ; <sup>c</sup>, significantly different between exercise at 40%  $\dot{V}O_{2max}$  and 70%  $\dot{V}O_{2max}$  ( $P<0.05$ ).

From the questionnaires taken before each main trial (Appendix 2), there was no difference between trials in sleeping time (Rest, 7±0hours; 40%  $\dot{V}O_{2max}$ , 7±0 hours; 70%  $\dot{V}O_{2max}$ , 7±0 hours, Student's *t*-test, all  $P>0.23$ ) and overnight fasting time (Rest, 13±0 hours; 40%  $\dot{V}O_{2max}$ , 14±0 hours; 70%  $\dot{V}O_{2max}$ , 13±0 hours, all  $P>0.38$ ). There was no difference in body weight, barometric pressure, humidity and temperature between trials. From Table 5.2 it can be seen that energy expenditure ( $P<0.01$ ), oxygen consumption ( $P<0.01$ ), total carbohydrate ( $P<0.01$ ) and fat oxidation ( $P<0.01$ ) during exercise at both moderate and high intensity were greater than rest trial. In addition, there was no significant difference in energy expenditure during moderate and high intensity exercise ( $P=0.5$ ).

### 5.3.2 Energy intake

Access to a buffet meal for a period of 30 minutes was provided one hour after rest or exercise. Participants consumed 13% and 17% more after moderate (40%  $\dot{V}O_2\text{max}$ ) and high intensity (70%  $\dot{V}O_2\text{max}$ ) exercise than during the resting trial, respectively, but these differences were not significant ( $P=0.23$  and  $P=0.15$  respectively). Relative energy intake and energy intake after subtraction of energy expenditure following rest was substantially greater than after high ( $P=0.04$ ) and moderate ( $P=0.02$ ) intensity exercise (Table 5.3).

	Rest	40%	70%
Energy intake (kcal)	1960±179	2210±164	2284±189
Carbohydrate (g)	289±30	345±37	365±44
Lipid (g)	75±15	78±8	76±10
Protein (g)	51±5	54±5	57±6
Relative energy intake (kcal)	1960±179	1443±169 <sup>a</sup>	1455±198 <sup>b</sup>
EI-EE (kcal)	1827±182	1306±168 <sup>a</sup>	1367±196 <sup>b</sup>

Table 5.3 Absolute and relative energy intake at lunch time on the trial days during a buffet meal. Values are means±SE (n=10). Relative energy intake (REI) = energy intake-(total energy cost of the exercise session-(exercise time x resting energy expenditure)). EI, energy intake during a buffet meal; EE, energy expended during laboratory-based exercise on the main trial day. 40%, exercise at 40%  $\dot{V}O_2\text{max}$ ; 70%, exercise at 70%  $\dot{V}O_2\text{max}$ . <sup>a</sup>, significantly different between rest and exercise at 40%  $\dot{V}O_2\text{max}$ ; <sup>b</sup>, significantly different between rest and exercise at 70%  $\dot{V}O_2\text{max}$  ( $P<0.05$ ).

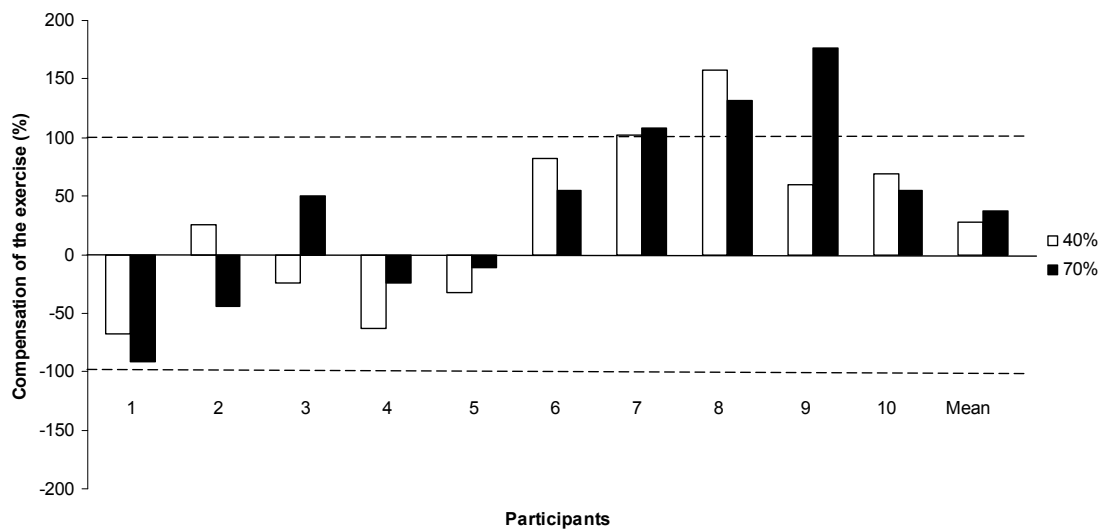


Figure 5.2 Compensation of exercise-induced energy expenditure during a buffet meal. Compensation =  $\left[ \frac{\text{energy intake during the buffet meal in each exercise trial} - \text{energy intake in control rest trial}}{\text{exercise cost of exercise above resting energy expenditure}} \right] \times 100$ . A value of 0% represents energy intake after exercise being equivalent to energy intake after the rest trial; a value of 100% represents energy intake after exercise being 100% greater than after rest and this is equivalent to the energy expended in the exercise session above rest; a value of -100% represents energy intake after rest being 100% greater than after exercise and is equivalent to the energy expended in the exercise session above rest (see general methods for more details). 40%, exercise at 40%  $\dot{V}O_2\text{max}$ ; 70%, exercise at 70%  $\dot{V}O_2\text{max}$ .

Figure 5.2 presents energy intake during the buffet meal expressed relative to energy expenditure and taking into account energy intake during the rest trial. This provides some information on the extent of immediate compensation of exercise-induced energy expenditure. The mean compensation of exercise in moderate and high intensity exercise trials was 28% and 38%, respectively ( $P=0.6$ ). Overall, there was relatively poor immediate compensation to exercise bouts (where 100% represents perfect compensation). There was individual variability in compensation to exercise. For example, participant seven shows perfect compensation in both exercise trials whereas participant one shows the opposite (this indicates that energy intake after rest trial is equal to energy expenditure in exercise trials).

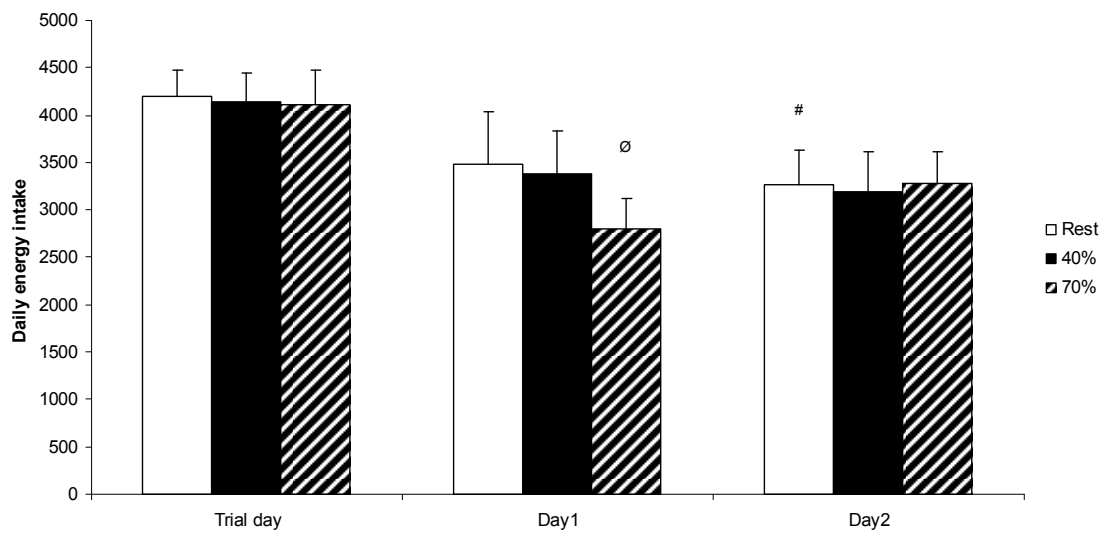


Figure 5.3 Energy intake (EI) in rest and exercise trials. EI from a buffet meal and weighed food and fluid records on each trial day; Day 1, one day after each main trial; Day 2, two days after each main trial. Values are mean $\pm$ SE (n=8). 40%, exercise at 40%  $\dot{V}O_2$ max; 70%, exercise at 70%  $\dot{V}O_2$ max. #, significantly different between trial day and day 1 or day 2 in rest trials; Ø, significantly different between trial day and day 1 or day 2 in exercise at 70%  $\dot{V}O_2$ max ( $P<0.05$ ).

Figure 5.3 shows energy intake estimated from three days weighed food and fluid records. According to weighed food and fluid records there was no difference in energy intake between trials. There was an effect of time for daily energy intake ( $P=0.01$ ). Daily energy intake decreased significantly on day one after 70%  $\dot{V}O_2$ max ( $P<0.01$ ) and on day two after rest trials ( $P=0.05$ ).

## 5.3.3 Subjective feelings of hunger and fullness

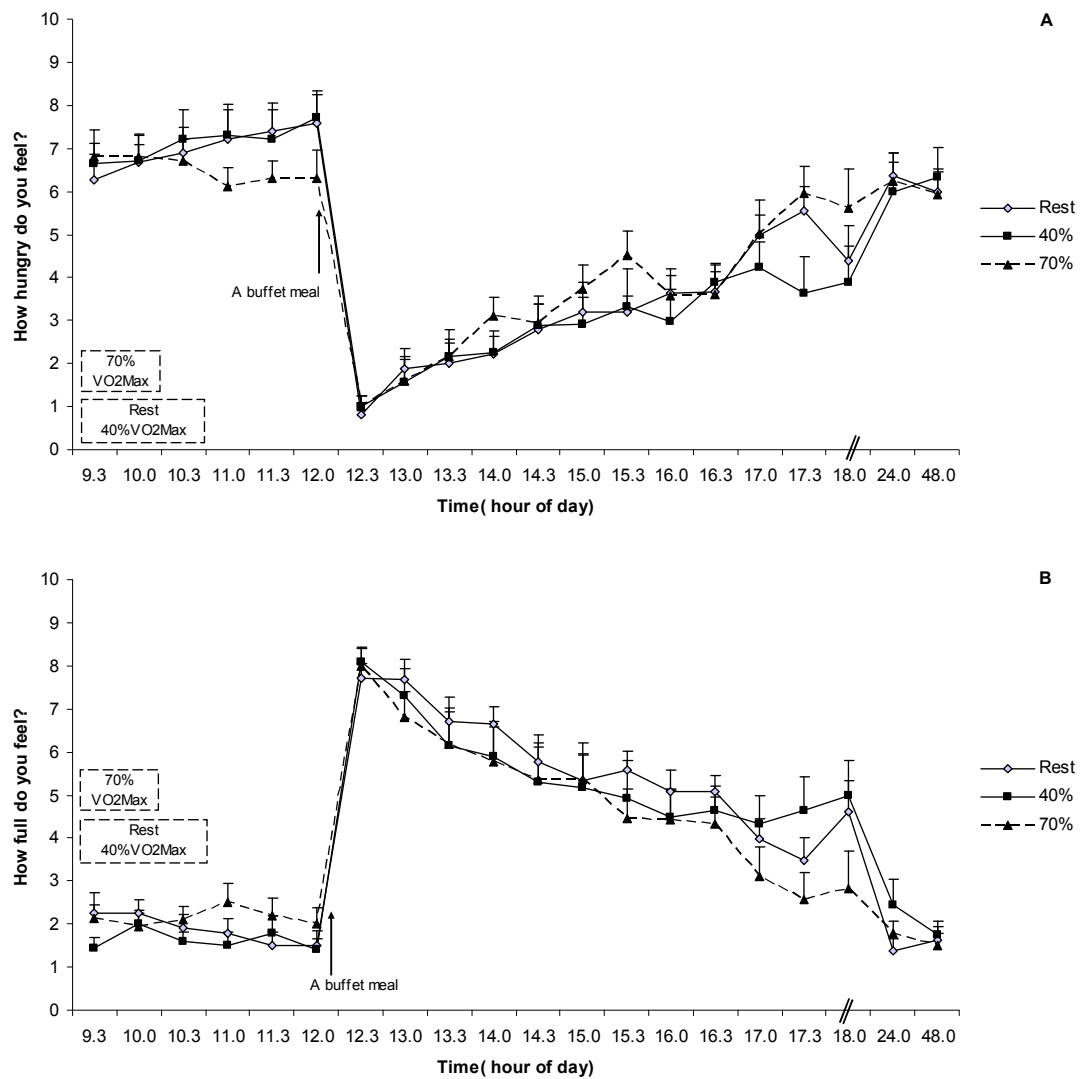


Figure 5.4 Subjective feelings of hunger and fullness using a 100 mm visual analogue scale (means $\pm$ SE) during exercise and rest trials (n=10). A, how hungry do you feel; B, how full do you feel. Data have been converted to a 0-10 scale for presentation. 40%, exercise at 40%  $\dot{V}O_2$ max; 70%, exercise at 70%  $\dot{V}O_2$ max.

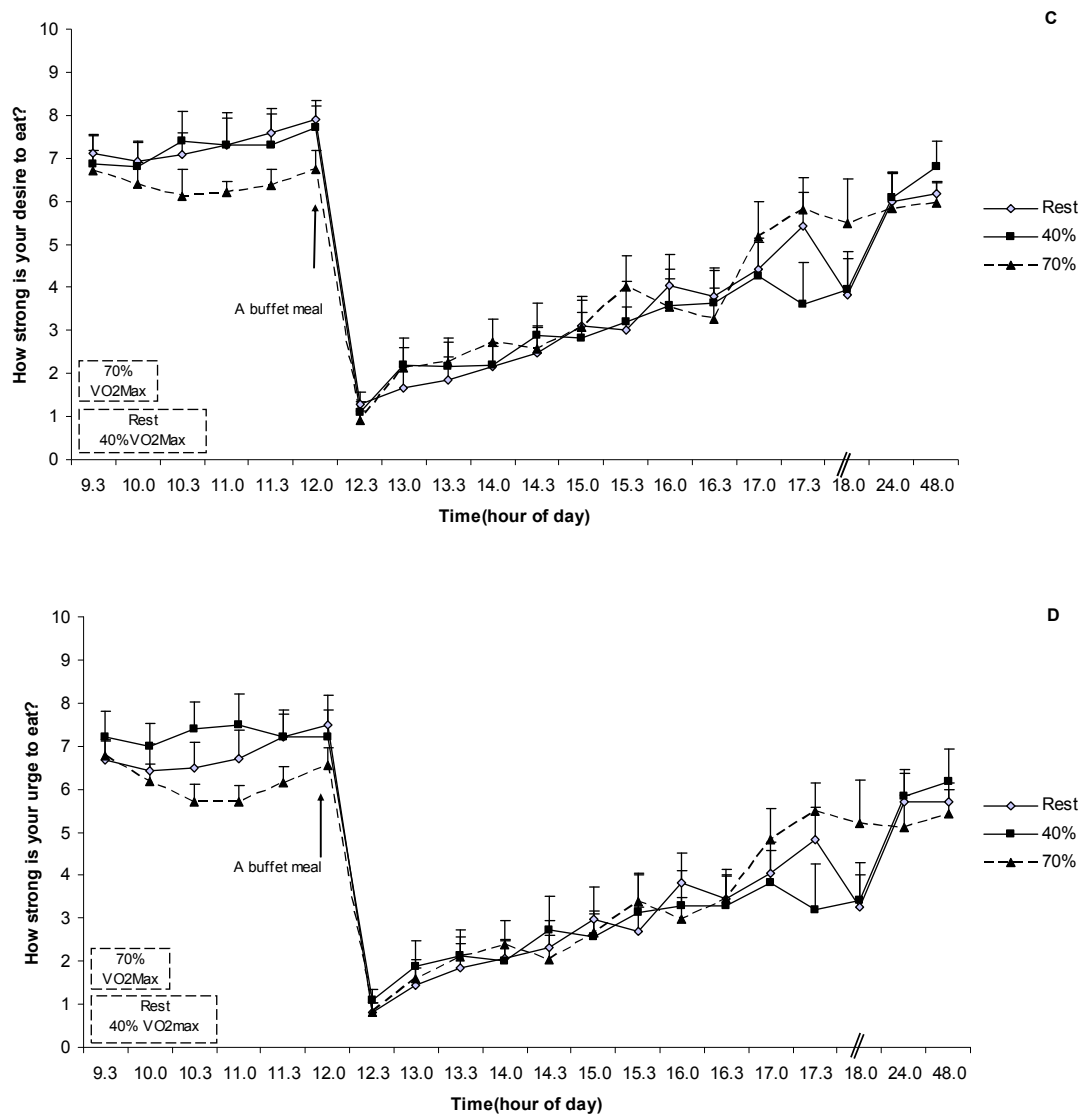


Figure 5.5 Subjective feelings of desire and urge to eat using a 100 mm visual analogue scale (means $\pm$ SE) during exercise and rest trials (n=10). A, how strong is your desire to eat; B, how strong is your urge to eat. Data have been converted to a 0-10 scale for presentation. 40%, exercise at 40%  $\dot{V}O_2$ max; 70%, exercise at 70%  $\dot{V}O_2$ max.

Subjective feelings of hunger and fullness were assessed every 30 minutes from 9.30 till 18.00 during each main trial, and then 24 and 48 hours following rest or exercise trials. As an overall trend, ratings of hunger, desire and urge to eat tended to be lower during and immediately after high intensity exercise but this effect was modest and not statistically significant. There was a time effect for hunger, fullness, desire and urge to eat, as a result of consumption of the buffet meal ( $P<0.01$ ). For clarity of presentation, these have not been depicted in the figures.

### 5.2.4 Physical activity energy expenditure

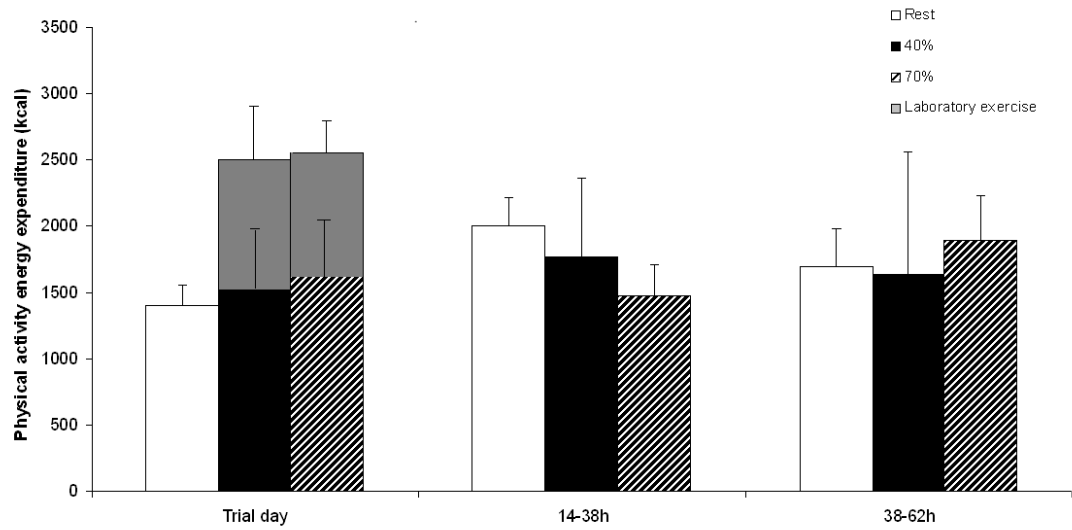


Figure 5.6 Physical activity energy expenditure (PAEE) in rest and exercise trials. Values are means $\pm$ SE (n=9). Trial day, rest or exercise; 14-38h, between 14-38 hours after each trial; 38-62h, between 38-62 hours after each trial. 40%, exercise at 40%  $\dot{V}O_2$ max; 70%, exercise at 70%  $\dot{V}O_2$ max.

There was there was no difference in PAEE between trials after correction for the energy expended during laboratory-based exercise on the main trial day (Figure 5.6).

## 5.3.5 Blood parameters

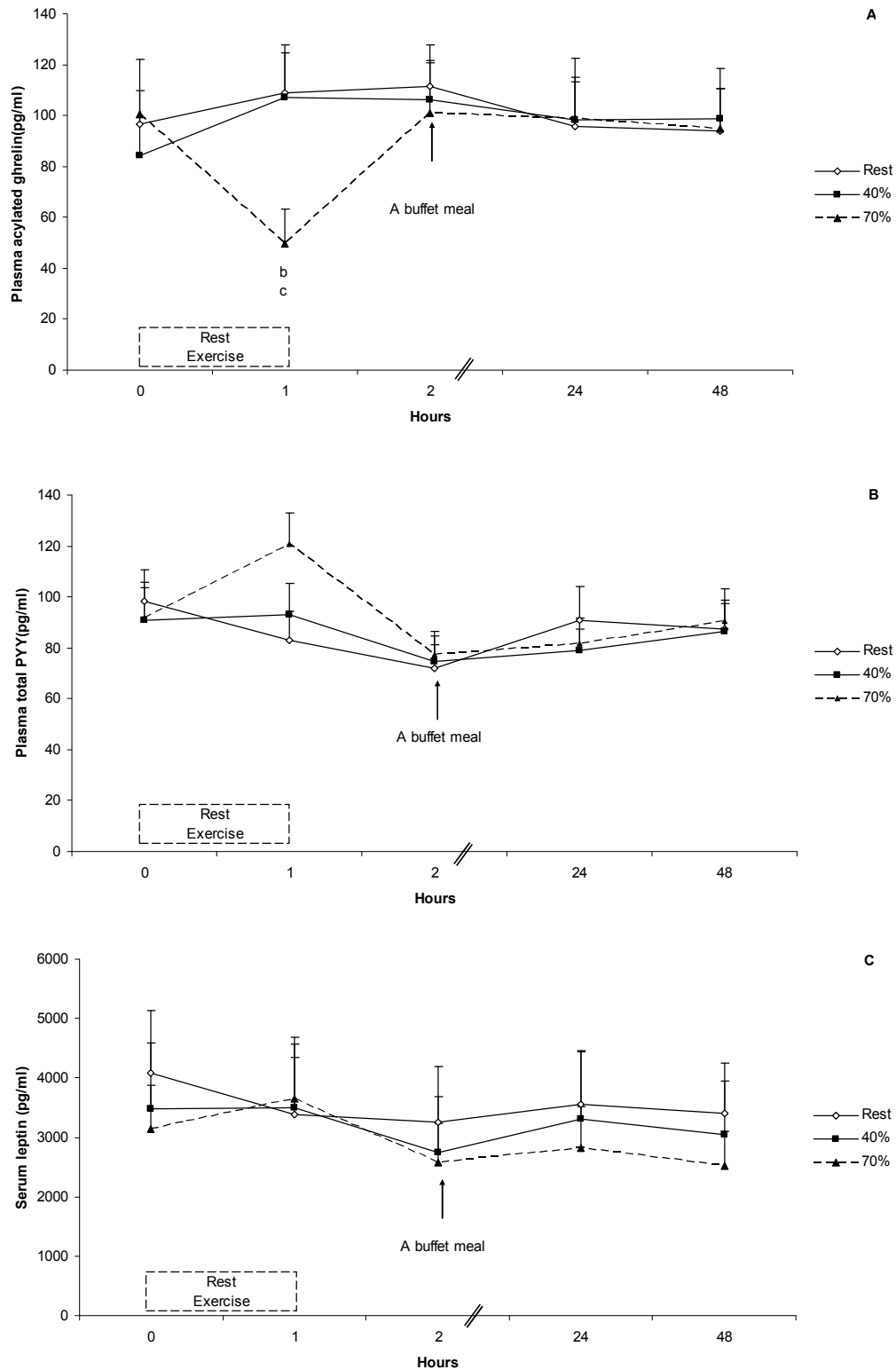


Figure 5.7 Acylated ghrelin (A), total peptide YY (PYY; B) and leptin (C) concentrations in rest and exercise trials. Values are means $\pm$ SE (n=10). 40%, exercise at 40%  $\dot{V}O_2$ max; 70%, exercise at 70%  $\dot{V}O_2$ max. <sup>b</sup>, significantly different between rest and exercise at 70%  $\dot{V}O_2$ max; <sup>c</sup>, significantly different between exercise at 40%  $\dot{V}O_2$ max and 70%  $\dot{V}O_2$ max ( $P<0.05$ ).



Figure 5.7 shows plasma acylated ghrelin, total PYY and leptin concentrations in exercise and rest trials. Two-way ANOVA showed a trial and time interaction ( $P < 0.01$ ) for plasma acylated ghrelin concentration. Plasma acylated ghrelin concentrations immediately following high intensity exercise were lower than after resting ( $P = 0.01$ ) and moderate intensity exercise ( $P = 0.01$ ).

Two-way ANOVA revealed a trend for the interaction between trial and time for plasma total PYY concentrations ( $P = 0.09$ ). Further analysis of these data showed that total PYY concentrations after exercise at 70%  $\dot{V}O_{2\max}$  were greater than 40%  $\dot{V}O_{2\max}$  ( $P = 0.05$ ) and rest ( $P = 0.04$ ). Serum leptin concentrations were not different between trials (Two-way ANOVA: Trial x time interaction,  $P = 0.17$ ).

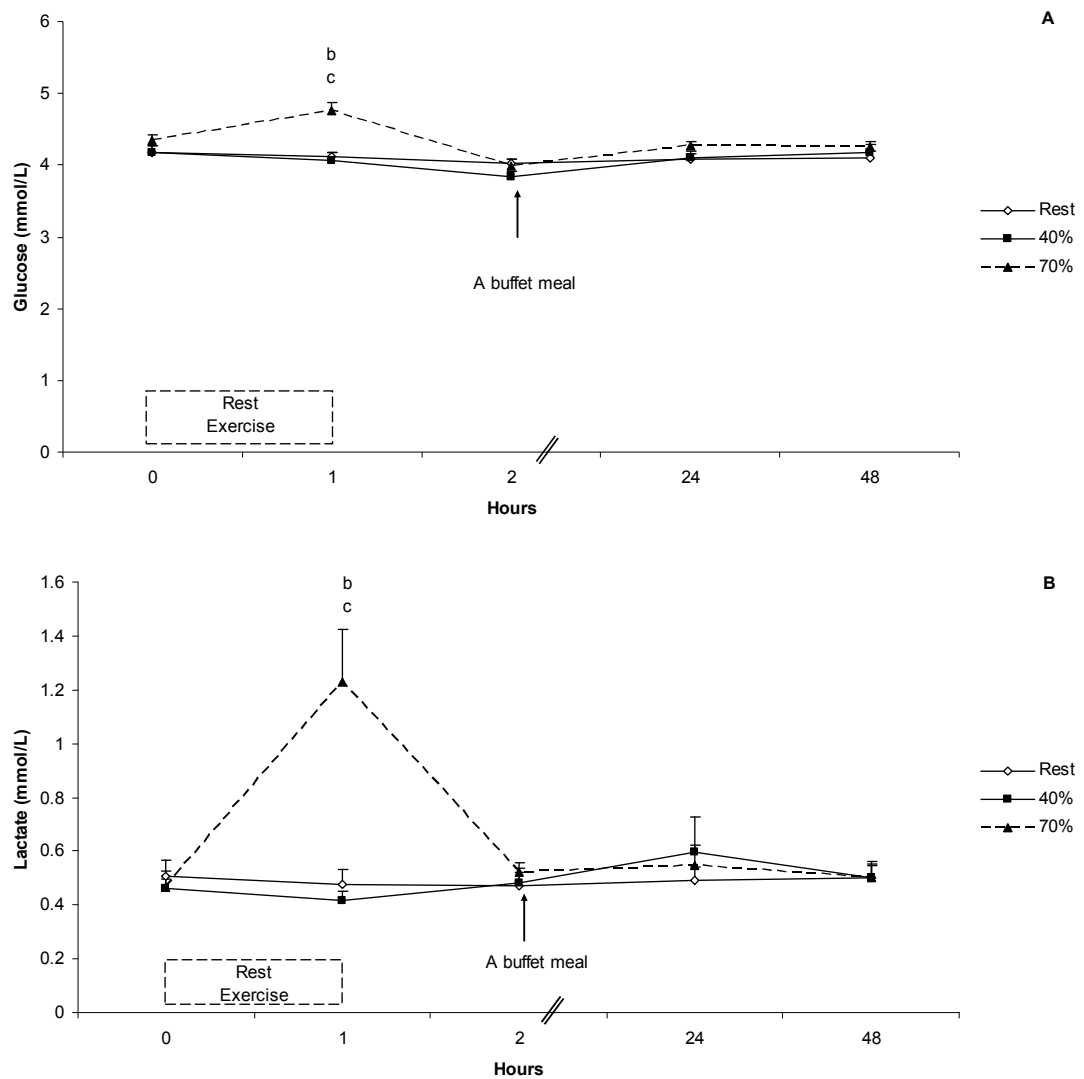


Figure 5.8 Glucose (A) and lactate (B) concentrations in rest and exercise trials. Values are means $\pm$ SE (n=10). 40%, exercise at 40%  $\dot{V}O_2$ max; 70%, exercise at 70%  $\dot{V}O_2$ max. <sup>b</sup>, significantly different between rest and exercise at 70%  $\dot{V}O_2$ max; <sup>c</sup>, significantly different between exercise at 40%  $\dot{V}O_2$ max and 70%  $\dot{V}O_2$ max ( $P<0.05$ ).

Figure 5.8 shows blood glucose and lactate concentrations in rest and exercise trials. Two-way ANOVA revealed a trial and time interaction for blood glucose ( $P=0.01$ ) and lactate ( $P<0.01$ ) concentrations. Blood glucose and lactate concentrations following high intensity exercise were higher than resting ( $P<0.01$  and  $P=0.01$  respectively) and moderate intensity exercise ( $P<0.01$  and  $P=0.01$  respectively)

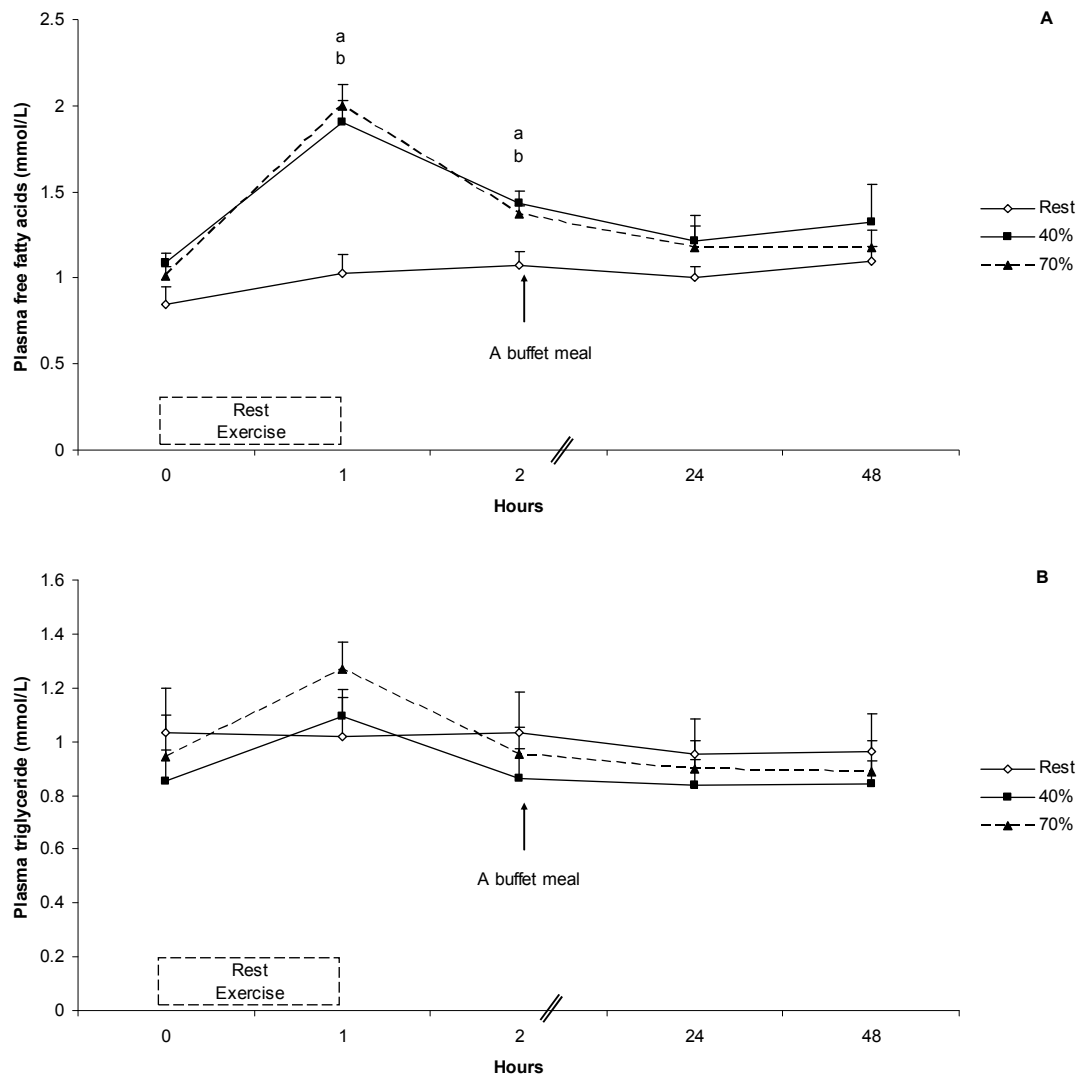


Figure 5.9 Plasma free fatty acids (A) and triglyceride (B) concentrations in rest and exercise trials. Values are means $\pm$ SE ( $n=10$ ). 40%, exercise at 40%  $\dot{V}O_2\text{max}$ ; 70%, exercise at 70%  $\dot{V}O_2\text{max}$ . <sup>a</sup>, significantly different between rest and exercise at 40%  $\dot{V}O_2\text{max}$ ; <sup>b</sup>, significantly different between rest and exercise at 70%  $\dot{V}O_2\text{max}$  ( $P<0.05$ ).

Figure 5.9 shows free fatty acids and triglyceride concentrations in each trial. Two-way ANOVA analysis indicated a trial and time interaction ( $P=0.01$ ) for plasma free fatty acids. There was a significant increase in plasma free fatty acids immediately and one hour following exercise at 40% ( $P<0.01$  and  $P=0.01$ ) and 70%  $\dot{V}O_2\text{max}$  ( $P<0.01$  and  $P=0.02$ ). Two-way ANOVA revealed a trial and time interaction ( $P=0.03$ ) for plasma triglyceride concentration, however, there were no differences in *post hoc* comparisons at specific time points.

## 5.4 Discussion

The primary objective of the present study was to investigate the impact of exercise on subsequent energy intake, appetite and related hormones and physical activity energy expenditure in young men who were habitually active. The main hypothesis was that active males would show good regulation of energy intake in response to exercise that is similar to their normal training. It was also anticipated that regulation of energy intake in response to high intensity exercise that was similar to their normal training might be better than in response to energy-matched moderate intensity exercise. However, we found no impact of high intensity exercise on absolute energy intake and appetite during a buffet meal one hour after exercise and also over the two days following exercise in active men compared with a rest trial. Furthermore, there was no difference in absolute energy intake and relative energy intake following moderate and high intensity exercise during a buffet meal. Interestingly, circulating plasma acylated ghrelin concentrations were suppressed while plasma total PYY concentrations tended to increase immediately following high intensity exercise compared with resting and moderate intensity exercise. However, this did not translate into a difference in absolute or relative energy intake or appetite either during a buffet meal or over the following two days.

In the present study, absolute energy intake during a buffet meal one hour after either high or moderate intensity exercise was not different to absolute energy intake during a rest (control) trial. These results are consistent with some previous studies (King et al. 1994; Imbeault et al. 1997; Erdmann et al. 2007) although other investigations have reported both an increase (Pomerleau et al. 2004) and decrease (Blundell et al. 1999; Kissileff et al. 1990) in energy intake during a buffet meal following high intensity exercise. Although there was no difference in absolute energy intake in a buffet meal between trials, the exercise-induced increase in energy expenditure meant that relative energy intake following moderate and high intensity exercise was considerably lower than the rest trial. This is consistent with earlier results (Chapter 4) and a previous investigation (Pomerleau et al. 2004). Additionally, there was no change in energy intake for two days after exercise and this is consistent with previous studies (Jamurtas et al. 2004; Chapter 4).

Moreover, there was no impact of moderate and high intensity exercise on subjective feelings of hunger and fullness in this study and this is consistent with previous studies (Pomerleau et al. 2004; Erdmann et al. 2007; Imbeault et al. 1997). In contrast, other studies demonstrated that subjective feelings of hunger was suppressed by high intensity exercise (King et al. 1994; Broom et al. 2007; Broom et al. 2009; Burns et al. 2007). It appears that there was no impact of either exercise bout on absolute energy intake one hour following exercise and also for two days according to weighed food and fluid records.

It should be noted that energy intake during the buffet meal in the main trials was considerable (approximately 50% typical daily energy intake). It is difficult to know whether this observation has an impact on the present findings. It is possible that because the food was freely available and unlimited, participants ate more than they would have done in other circumstances. If this was more pronounced in the rest trial this could have masked any impact from exercise. Alternatively, it is possible that because participants arrived in a fasted state that energy intake during the buffet meal represents an attempt to 'catch up'. Interestingly, there was considerable individual variability in absolute energy intake and compensation to exercise which is similar to findings in sedentary men (Chapter 4). Therefore, even though the participants were active and familiar with exercise, there was no evidence that this was associated with better regulation of energy intake either in the short-term (during a buffet meal) or in the medium-term (over the following few days) than their sedentary counterparts (Chapter 4). It was speculated that we might see better regulation of energy intake in response to familiar exercise (i.e., the high intensity trial) but there was no evidence that this was the case.

In previous studies (Chapters 3 and 4), it was found that physical activity energy expenditure (PAEE) after moderate intensity exercise was increased for ~62 hours in sedentary males. In the present study, this was not the case and PAEE over the two days after either the high intensity or moderate intensity exercise trials was not different to the corresponding rest trial. This result is consistent with some studies showing that there is no change in physical activity energy expenditure after two sessions of cycling in active males (Seale et al. 1996; King et al. 1997). Whereas in sedentary men, non-

prescribed PAEE was increased chronically after an eight months exercise intervention (Hollowell et al. 2009). It is possible that when physical activity is low that structured exercise actually has a positive impact on non-exercise PAEE (Chapter 4) whereas when physical activity is high and regular (the present study) that no such affect is observed.

Several hormones have been proposed to play a role in the regulation of energy intake and physical activity energy expenditure (e.g., leptin, total PYY and acylated ghrelin). In the present study, circulating plasma acylated ghrelin concentrations were lower immediately following high intensity exercise (but not moderate intensity exercise). This effect of high intensity exercise has been found in previous studies (Broom et al. 2007; Broom et al. 2009) although there was no change in plasma acylated ghrelin concentrations following high intensity exercise in sedentary males (Chapter 3). It is possible that either activity status or the fact that overall energy expenditure (rate and total) would have been lower in sedentary men (Chapters 3 and 4) partly explains this finding. It appears that a change in circulating plasma acylated ghrelin concentrations might depend not only on the intensity of exercise but also on the total energy expended during exercise and the physical activity level of the participants. In this study, the impact of high intensity exercise on circulating plasma acylated ghrelin concentrations was short-lived and present only immediately after exercise. Blood glucose concentrations after high intensity exercise were higher than moderate intensity exercise and resting in this study. This is consistent with a previous study (Broom et al. 2007). Acylated ghrelin administration leads to decrease in the concentration of glucose and an increase in glucose response after a meal in young men (Broglia et al. 2008). It is possible that suppressed in acylated ghrelin in the present study might be related to elevated blood glucose concentration. However, this is speculation the numerous other parameters would also be different between high and moderate intensity exercise.

Plasma total PYY concentrations tended to increase one hour following high intensity exercise but there was no change in total PYY concentrations after moderate intensity exercise in the present study. One recent study showed that plasma total PYY concentrations were elevated following high intensity exercise and remained elevated after a meal (Broom et al. 2009). Another study demonstrated that moderate intensity

exercise has a small impact on postprandial total PYY concentrations (Martin et al. 2007). In the present study, as was the case for acylated ghrelin, the increase in total PYY concentrations following high intensity exercise was short-lived. After high intensity exercise, both acylated ghrelin and PYY were no longer different one hour after exercise and at the time of the buffet meal. Therefore, notwithstanding the limitations associated with the interpretation of the energy intake data discussed above, it appears that the short-term changes in acylated ghrelin and PYY do not translate into altered energy intake.

Serum leptin concentrations did not differ between trials in the present study. These findings are consistent with previous studies which show that leptin concentration is not affected by a single bout of exercise (Perusse et al. 1997; Racette et al. 1997; Esssing et al. 2000; Weltman et al. 2000) although some studies have shown that serum leptin concentrations fall after vigorous intensity exercise (Landt et al. 1997; Duclos et al. 1999; Koistinen et al. 1998).

In addition to the concerns regarding energy intake during the buffet meal discussed above, there are some other limitations to the present study. There was no evidence of different compensation to different intensity exercise during a buffet meal (i.e., participants who compensated well did do irrespective of exercise intensity). However, there was inter-individual variability of compensation for energy intake during a buffet meal following exercise in active males in this study. This is consistent with Chapter 4 where there was variability of compensation after exercise in sedentary males. It appears that energy intake or compensation of exercise might not be associated with physical activity status. We might not expect full or even consistent compensation during a single meal in the post exercise period. If there is compensation over the days after exercise then it is possible that weighed food and fluid records are not sensitive enough to capture potentially small differences over this time

In summary, energy intake, appetite and physical activity energy expenditure did not change following either high intensity or moderate intensity exercise in active males. The impact of acute exercise on plasma acylated ghrelin and total PYY was short-lived and did not appear to alter energy intake and appetite during a subsequent buffet meal.

We do not know whether energy intake during a buffet meal would have been different if this coincided with altered hormone concentrations. It is possible that we need to strategically place the exercise bout at key times relative to food consumption in order to capitalise on the impact of acute exercise on appetite hormones. For example, future research might investigate the impact of exercise on appetite, energy hormones and energy intake in the hours following a meal.



## CHAPTER 6

### THE EFFECT OF ACUTE EXERCISE AFTER SHORT-TERM FOOD RESTRICTION ON ENERGY BALANCE HORMONES IN MALES AGED BETWEEN 18 AND 45 YEARS

#### 6.1 Introduction

In previous research (Chapter 5) it was shown that high intensity exercise (70%  $\dot{V}O_2\text{max}$ ) after an overnight fast results in decreased circulating acylated ghrelin concentration whereas total PYY concentration tended to increase immediately after exercise. Additionally, there was no change in post-exercise energy intake in a buffet meal after high intensity exercise in the fasted state. One study has shown that high intensity exercise followed by a test meal results in increased total PYY concentration but there was no change in acylated ghrelin concentration in well-trained participants (Broom et al. 2009). Another study demonstrated that intermittent cycling exercise in a fed state leads to an increase in postprandial total PYY and PP concentrations; however, there was no change in total ghrelin concentration (Martin et al. 2007). It is possible that high intensity exercise might change energy balance hormones in response to a meal and perhaps enhance satiety hormones (e.g., PYY).

Exercise is often combined with food restriction (dieting) to reduce weight. The effects of exercise superimposed on food restriction on energy balance hormones are unknown because participants in previous studies typically consumed their normal diet (Martin et al. 2007; Broom et al. 2009). A reduction in energy intake (dieting) will inevitably lead to feelings of hunger (Doucet et al. 2004) and this is partly because of changes in the hormones that influence appetite (Leidy et al. 2007). There may also be lower satisfaction after eating a meal. It has been demonstrated that some satiety hormone such as PYY and leptin are lower even after short-term food restriction (Doucet et al. 2004; Chan et al. 2004; Mars et al. 2005). However, little is known about the response to eating after a few days of food restriction. It is plausible that both fasting and postprandial energy balance hormones might be altered after a few days of food restriction and that this could influence the desire to eat.

The present study was designed to examine whether exercise influences postprandial energy balance hormones when superimposed on a period of food restriction (to mimic dieting). In pilot work, we have shown that total PYY concentrations decrease after three days of 50% reduced food intake in four participants (Appendix 8). Additionally, high intensity exercise after eating leads to an increase in total PYY and PP concentrations in two participants (Appendix 8). Therefore, the main objective of this study is investigate the impact of high intensity exercise in the fed state after three days of 50% food restriction on circulating energy balance hormones, post-exercise energy intake and physical activity energy expenditure. The main hypothesis is that postprandial circulating total PYY, PP and leptin concentration will increase more after exercise than rest whereas acylated ghrelin concentration will be unaffected.

## 6.2 Methods

### 6.2.1 Participant recruitment and eligibility

	Mean±SE
Age (years)	32±2
Body mass (kg)	77±3
Height (cm)	178±2
BMI (kg/m <sup>2</sup> )	24±1
Waist (cm)	83±2
Hip (cm)	99±2
Waist/Hip ratio	0.8±0
Total percent body fat (%)	17±2
Total fat mass (kg)	13±2
Total lean mass (kg)	59±2
Blood pressure: systolic / diastolic (mmHg)	128±3/77±4
Resting heart rate (beats /min)	58±3
$\dot{V}O_{2\max}$ (ml/kg/min)	60±2
Physical activity level (PAL)	2.0±0.1
Activity > 4.8 METs (minutes/day)	59±16

Table 6.1 Participants characteristics. Values are means  $\pm$  SE (n=11). BMI, body mass index;  $\dot{V}O_{2\max}$ , maximal oxygen consumption. Body composition determined by dual-energy x-ray absorptiometry (DEXA). Physical activity level (PAL) and metabolic equivalent (METs) estimated from physical activity monitor (Actiheart) worn for seven days.

Eleven active males aged between 18 and 45 years (Table 6.1) participated in this study, which had approval from the Local Ethics Committee. In order to assess eligibility, participants completed a health history questionnaire and a Physical Activity Readiness Questionnaire (PAR-Q). The participants were non-smokers with a body mass index of less than 30 kg/m<sup>2</sup>. Furthermore, their body weight had not changed more than 5 kg in the last six months. In order to take part, volunteers had to be regularly active and to be eligible to take part they reported that they ran (60 minutes) at least once a week (from self report, Appendix 1). The participants provided written informed consent.

Participants wore a physical activity monitor for seven days in order to assess physical activity energy expenditure. As shown in Table 6.1, the participants were very active with a physical activity level (PAL) greater than 1.9 (Brooks et al. 2004). Actiheart data were analysed in order to confirm that they did at least 30 minutes of physical activity above 7.2 METs; defined as hard physical activity (Pollock et al. 1998). Additionally, these data were also analysed to determine the amount of physical activity > 4.8 METs (moderate intensity physical activity).

### 6.2.2 Experimental design

This was a randomised cross-over design with two main trials separated by at least 14 days (Exercise *versus* Rest).

#### *3 days before a main trial*

Participants came to the laboratory between 8.00 and 8.30 a.m. after an overnight fast and a baseline blood sample (15 ml) was taken from a forearm vein. Then, in both main trials, participants were asked to follow a set diet over the three following days. This was based on their normal diet and was approximately 50% less than their normal diet (based on a seven days weighed food and fluid record). Participants were also asked to maintain their normal physical activity after a baseline sample. Physical activity energy expenditure was monitored (Actiheart, Cambridge Neurotechnology Ltd. Cambridge).

#### *Main trial day*

On one occasion participants performed a standardised bout of exercise and on another occasion participants rested in the laboratory for the same period of time (Figure 6.1). Each trial was separated by at least two weeks. In the exercise trial, participants ran on a treadmill at 70% of  $\dot{V}O_{2\max}$  for 60 minutes.

Participants came to the laboratory at approximately 8.00 a.m. after an overnight fast. On arrival at the laboratory, anaesthetic cream was applied 30 minutes before a cannula was inserted into a vein in their arm. In order to monitor the responses to exercise or rest, a blood sample (15 ml) was taken at baseline and every 30 minutes for the following four hours. Participants were asked to complete subjective feelings of hunger

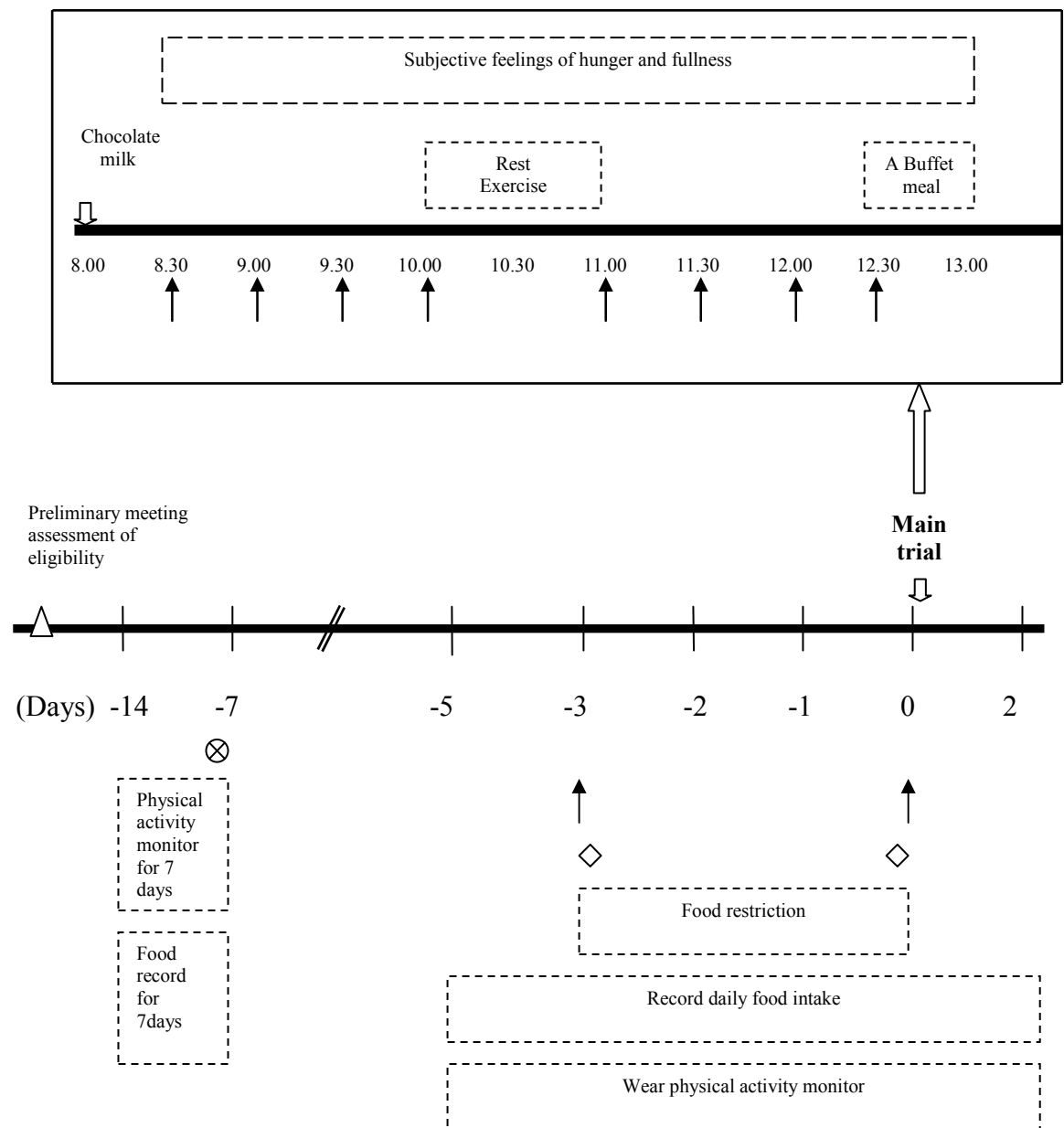
and fullness (100 visual analogue scales) over the same period of time (General Methods). A standard breakfast (chocolate milk) was provided after the baseline sample had been collected. 100 ml of chocolate milk contained 4.2 g protein, 11.7 g carbohydrate, 11.4 g sugar, 3.8 g fat (Sainsbury's, United Kingdom). This breakfast meal was based on that provided in a previous study (Martin et al. 2007).

### **6.2.3 Energy intake**

A buffet meal was provided 1.5 hours after each rest or exercise trials (details of the buffet meal are in general methods and Appendix 5). Participants were given 30 minutes (12.30 to 13.00) to consume food from the buffet. The buffet was designed to be similar to typical lunchtime foods and was the same as used in Chapters 4 and 5 (General Methods and Appendix 5). Additionally, energy intake outside the laboratory was assessed. To do this, participants weighed and recorded their daily food intake on the main trial day and for two days after each main trial. Dietary composition was analysed using computer software (COMP-EAT version 5, Carlson Bengston Consultants Limited, London).

### **6.2.4 Subjective feelings of hunger and fullness**

As explained in the general methods (Chapter 2), subjective feelings of hunger and fullness (Appendix 3) were assessed using 100 mm visual analogue scales (VAS). Participants indicated their appetite by marking on the line. In addition, subjective feelings of hunger and fullness were also assessed at the baseline, after food restriction and every 30 minutes on main trial days.



- ↑ Blood sample  
 ◇ Subjective feelings of hunger and fullness  
 ⊗ Preliminary measurement
- Maximum oxygen consumption
  - Blood pressure
  - Resting heart rate
  - Body composition

Figure 6.1 Study protocol. Main trial represents either rest or exercise (70%  $\dot{V}O_2\text{max}$ ) in a randomized order with at least two weeks between trials.

### **6.2.5 Energy expenditure and physical activity energy expenditure**

During rest or exercise bouts on each main trial day, expired gas was collected for one minute every ten minutes for each exercise trial and for five minutes in the rest trial in order to estimate energy expenditure using indirect calorimetry. Further details on these measurements can be found in the general methods (Chapter 2). To examine physical activity energy expenditure outside the laboratory, participants wore a physical activity monitor (Actiheart, Cambridge Neurotechnology Ltd., Cambridge) during food restriction, the main trial day and also two days following each main trial (General methods, Chapter 2). Physical activity energy expenditure (PAEE) is the energy expended above rest after subtraction of the energy spent during exercise in the laboratory.

### **6.2.6 Energy intake and physical activity energy expenditure control**

Participants asked to avoid alcohol and caffeine for 48 hours before sampling at baseline and in each main trial. Participants self-reported their normal daily food intake for seven days. Participants were then asked to consume 50% of their normal daily food intake for three days after -3 days sample. Participants were asked to maintain their normal physical activity during food restriction period after the baseline sample. Participants wore physical activity monitor and were asked to avoid strenuous exercise for one day before baseline and on the main trial day in order to control lifestyle and physical activity patterns in this period. Average physical activity energy expenditure (PAEE) for two days prior to each trial did not differ significantly between trials (Rest,  $1414 \pm 235$  kcal; Exercise,  $1167 \pm 129$  kcal,  $P=0.28$ ) before food restriction. In addition, PAEE for three days during food restriction did not differ significantly on day one (Rest,  $1446 \pm 202$  kcal; Exercise,  $1422 \pm 211$  kcal,  $P=0.83$ ).

### 6.2.7 Statistical analysis

Sample size was estimated using previous work that shows that the level of total PYY following aerobic exercise is 75 pg/ml greater than a control trial where the standard deviation of the difference is 83 pg/ml (Broom et al. 2009). Therefore, to detect a mean difference of 95 pg/ml in the present trial requires a estimated sample size of 10 and allowing 1 subject dropout (80% power and 5% alpha).

All data were analysed using Statistical software (SPSS 14.0, United States). Two-way repeated measures ANOVA and *t*-tests were used to analyse differences for total PYY, PP, leptin, acylated ghrelin, blood glucose, blood lactate concentrations and subjective feelings of hunger and fullness between trials over period of time. Paired *t*- tests were used for *post hoc* comparisons with a Bonferroni method. Paired *t*-tests were used to examine changing over time within each trial. Statistical significance was accepted at the 5% level ( $P < 0.05$ ). Data are presented as means $\pm$ SE.



### 6.3 Results

#### 6.3.1 Blood parameters

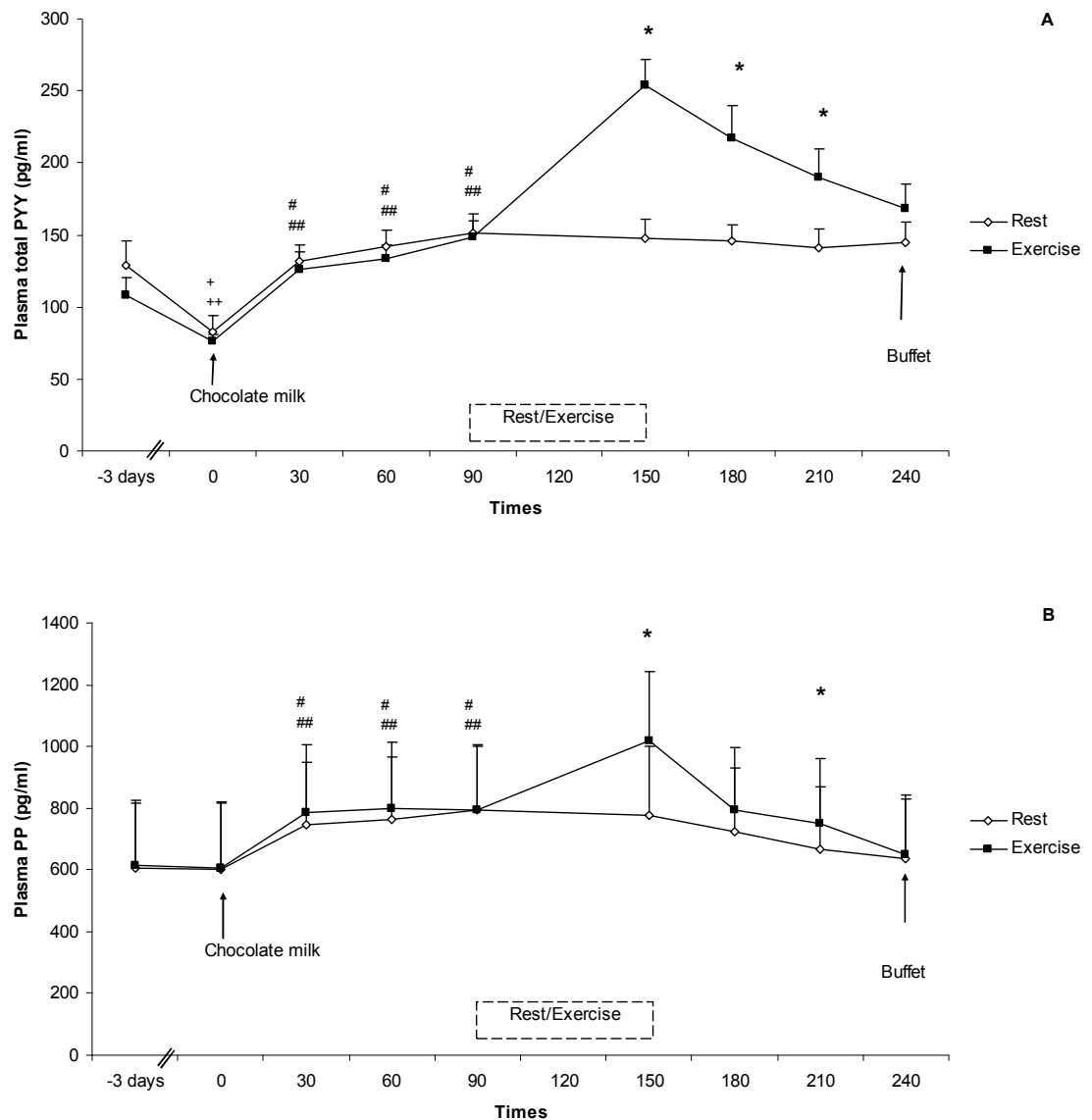


Figure 6.2 Total peptide YY (PYY; A) and pancreatic polypeptide (PP; B) concentrations in rest and exercise trials. Values are means $\pm$ SE (n=11). \*, significantly different between rest and exercise trials; +, significantly different between before (-3 days) and after (0 minute) food restriction in rest trials; ++, significantly different between before (-3 days) and after (0 minute) food restriction in exercise trials; #, significantly different between before (0 minute) and after (30, 60 and 90 minutes) chocolate milk in rest trials; ##, significantly different between before (0 minute) and after (30, 60 and 90 minutes) chocolate milk in exercise trials ( $P<0.05$ ).

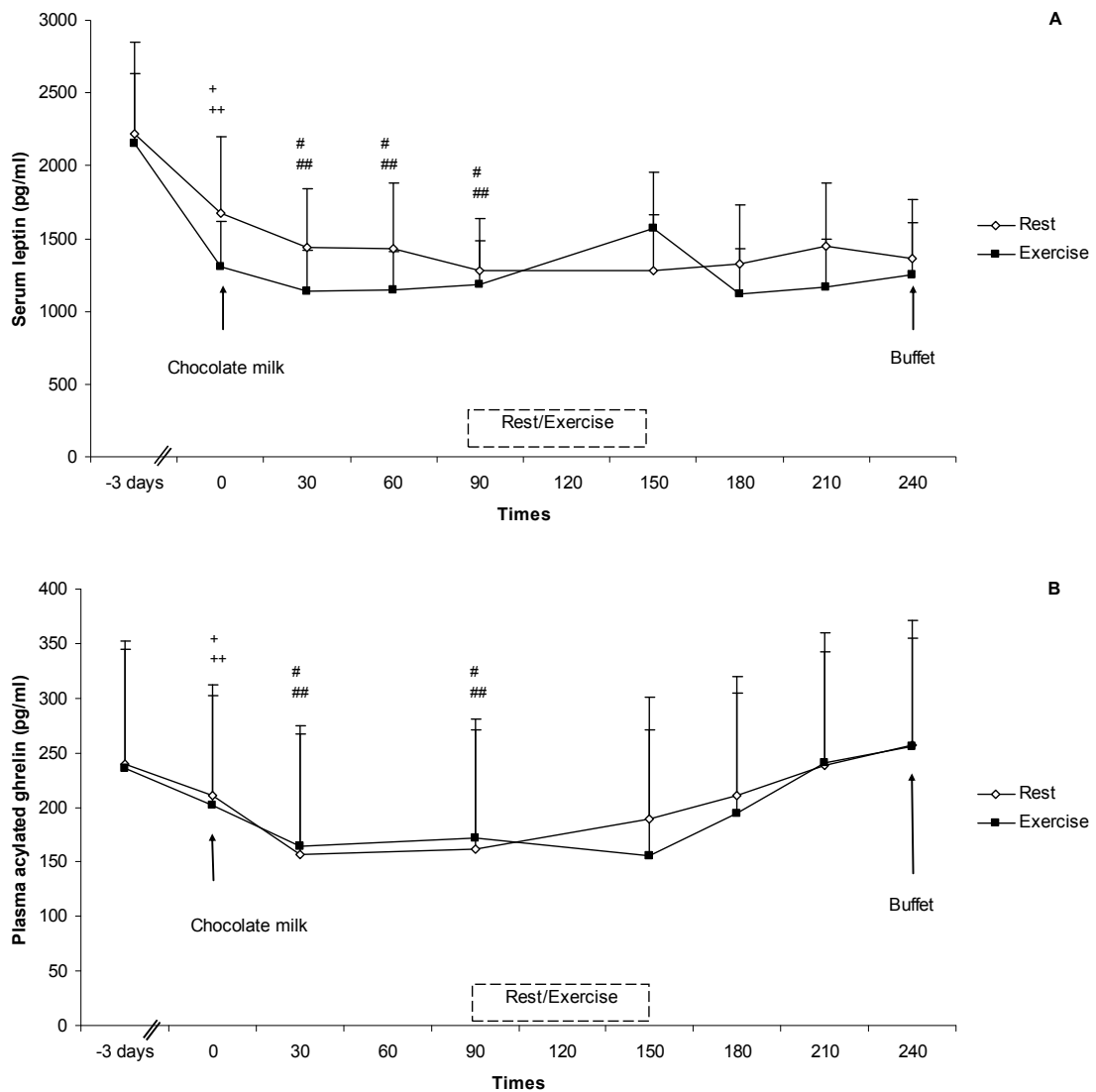


Figure 6.3 Leptin (A) and acylated ghrelin (B) concentrations in rest and exercise trials. Values are means $\pm$ SE (n=11). <sup>+</sup>, significantly different between before (-3 days) and after (0 minute) food restriction in rest trials; <sup>++</sup>, significantly different between before (-3 days) and after (0 minute) food restriction in exercise trials; <sup>#</sup>, significantly different between before (0 minute) and after (30, 60 and 90 minutes) chocolate milk in rest trials; <sup>##</sup>, significantly different between before (0 minute) and after (30, 60 and 90 minutes) chocolate milk in exercise trials ( $P<0.05$ ).

Figures 6.2 and 6.3 show the concentrations of selected hormones between rest and exercise trials. There was no significant difference in the concentrations of total PYY, PP, leptin, and acylated ghrelin at -3 days, before chocolate milk (breakfast) and immediately before rest or exercise (all  $P>0.13$ ). Fasting total peptide PYY ( $P<0.01$ ), leptin ( $P<0.01$ ) and acylated ghrelin ( $P=0.01$ ) concentrations decreased significantly after three days of food restriction but there was no change in pancreatic polypeptide (PP) concentration ( $P=0.66$ ). Postprandial total PYY and PP concentrations rose

significantly while postprandial leptin and acylated ghrelin concentrations decreased significantly 30, 60, 60 minutes following the chocolate milk (*t*-test,  $P<0.01$ ).

Two-way ANOVA revealed a trial and time interaction ( $P<0.01$ ) for postprandial total PYY concentrations between trials. *Post hoc* analysis indicated that exercise caused an increase in postprandial total PYY concentrations immediately ( $P<0.01$ ), 30 ( $P<0.01$ ) and 60 ( $P<0.01$ ) minutes following exercise.

Circulating postprandial PP concentration in the exercise trial was higher than rest at immediately ( $P=0.05$ ) and 60 ( $P=0.05$ ) minutes following exercise (Two-way ANOVA: Trial x time interaction,  $P=0.01$ ). However, there was no difference in plasma acylated ghrelin and serum leptin concentrations between rest and exercise trials

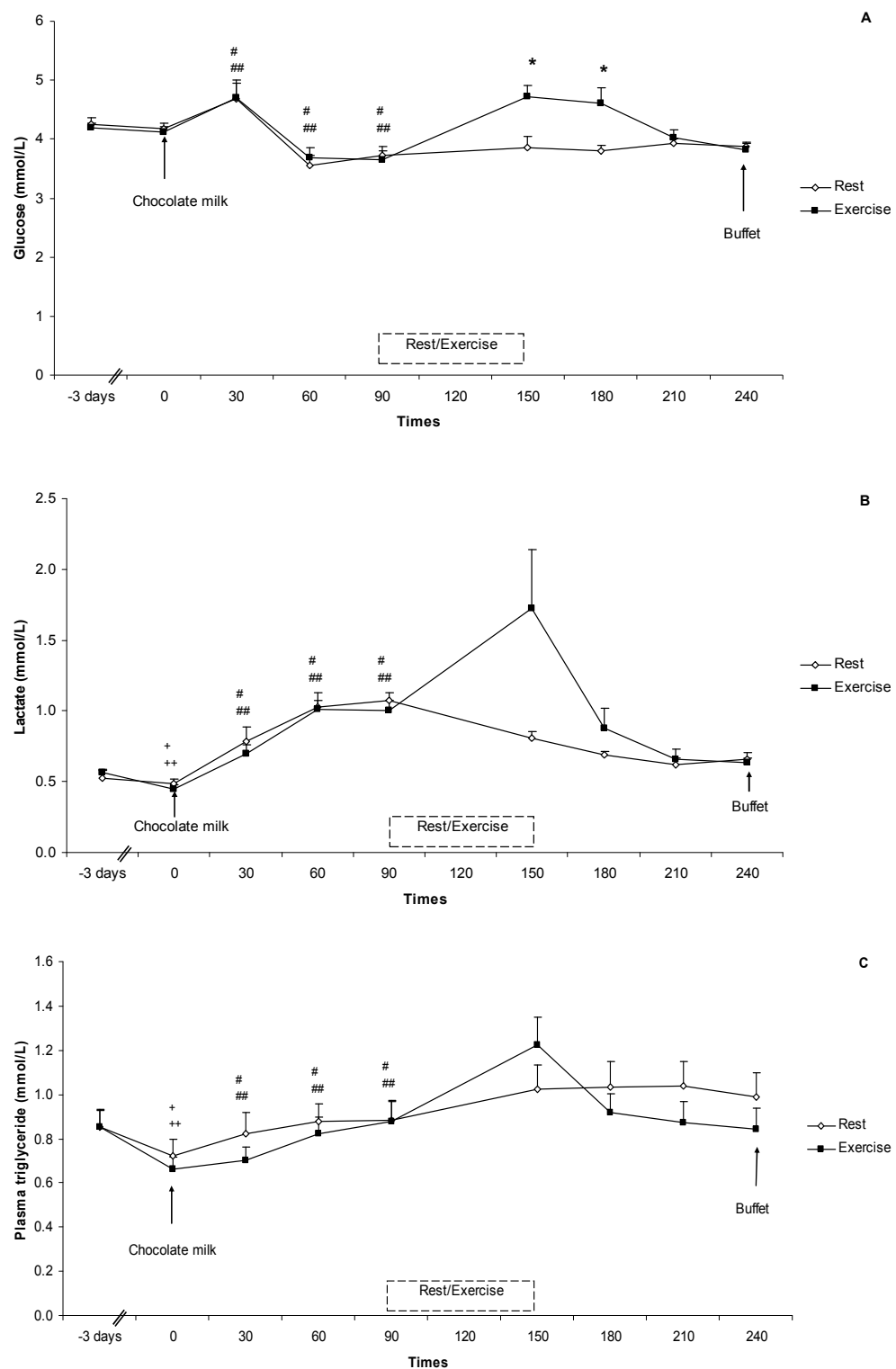


Figure 6.4 Glucose (A), lactate (B), triglyceride (C) concentrations in rest and exercise trials. Values are means $\pm$ SE (n=11). \*, significantly different between rest and exercise trials; +, significantly different between before (-3 days) and after (0 minute) food restriction in rest trials; ++, significantly different between before (-3 days) and after (0 minute) food restriction in exercise trials; #, significantly different between before (0 minute) and after (30, 60 and 90 minutes) chocolate milk in rest trials; ##, significantly different between before (0 minute) and after (30, 60 and 90 minutes) chocolate milk in exercise trials ( $P<0.05$ ).

Figure 6.4 Blood glucose, lactate and triglyceride concentrations did not differ significantly between trials at -3 days, three days after food restriction and before rest or exercise (all  $P>0.2$ ). Blood lactate ( $P=0.01$ ) and triglyceride ( $P<0.01$ ) concentrations decreased significantly after three days 50% of food restriction but there was no change in blood glucose concentrations ( $P=0.21$ ). Postprandial blood lactate and triglyceride concentrations increased significantly 30, 60, 90 minutes following the breakfast meal ( $P<0.01$ ). Postprandial blood glucose concentrations increased significantly after 30 minutes and decreased dramatically 60 minutes following the breakfast meal ( $P<0.01$ ).

Postprandial blood glucose concentrations increased significantly immediately ( $P=0.04$ ) and 30 minutes ( $P=0.04$ ) following exercise (Two-way ANOVA: Trial x time interaction,  $P<0.01$ ). Two-way ANOVA revealed that there was an interaction between trial and time for blood lactate ( $P=0.03$ ) and blood triglyceride ( $P=0.01$ ) concentrations but there was no difference after *Post hoc* tests were adjusted using the Bonferroni method.

**6.3.2 Trial data**

	Rest	Exercise
Environmental		
- Barometric pressure (mmHg)	741±0	741±0
- Temperature (°C)	20±0	20±0
- Humidity (%)	42±2	44±2
% $\dot{V}O_{2\max}$	8±0	72±1*
Energy expenditure (kcal)	112±7	931±35*
Energy expenditure/BW (kcal/kg)	2±0	12±0*
Total carbohydrate oxidation (g)	13±3	149±14*
Total fat oxidation (g)	6±1	35±6*

Table 6.2 Trial data in rest and exercise trials. Values are means±SE (n=11). %  $\dot{V}O_{2\max}$ , percent of maximum oxygen consumption every ten minutes. \*, significantly different between rest and exercise at 70%  $\dot{V}O_{2\max}$  ( $P<0.05$ ).

From the questionnaires prior to the main trial days (Appendix 2), Student *t*-test showed that sleeping time (Rest, 7±3 hours; exercise, 7±3hours;  $P=0.13$ ) and overnight fasting time (Rest, 12±0 hours; exercise, 12±0 hours;  $P=0.87$ ) did not differ between rest and exercise trials. Mean body weight before at baseline before food restriction were 76.5 kg (rest) and 76.6 kg (exercise) and did not differ significantly between trials ( $P=0.63$ ). Following 50% food restriction for three days, body weight was considerably lower than baseline (Rest, 75.1 kg; Exercise, 74.8 kg,  $P<0.01$ ). However, body weight between trials did not differ significantly after three days food restriction ( $P=0.47$ ). As shown in Table 6.2, oxygen consumption, energy expended, total carbohydrate oxidation and total fat oxidation during the exercise trial were greater than during the rest trial ( $P<0.01$ ).

### 6.3.3 Energy intake

	Rest	Exercise
Energy intake (kcal)	1736±274	1623±254
Carbohydrate (g)	252±47	262±49
Lipid (g)	67±12	51±8
Protein (g)	47±6	46±6
Relative energy intake (kcal)	1736±274	786±246*
EI-EE (kcal)	1624±275	693±250*

Table 6.3 Absolute and relative energy intake at lunch time on the trial days during a buffet meal. Values are means±SE (n=11). Relative energy intake (REI) = energy intake-(total energy cost of the exercise session-(exercise time x resting energy expenditure)). EI, energy intake during a buffet meal; EE, energy expended during laboratory-based exercise on the main trial day. \*, significantly different between rest and exercise at 70%  $\dot{V}O_2$ max ( $P<0.05$ ).

Table 6.3 shows absolute and relative energy intake between trials. A buffet meal was provided 1.5 hours after rest or exercise. Although mean absolute energy intake (113 kcal) and lipid intake (16 g) following exercise were considerably less than following rest, there was no statistical difference in absolute energy intake between trials ( $P=0.43$ ). However, there was significant decrease in relative energy intake after exercise ( $P<0.01$ ).

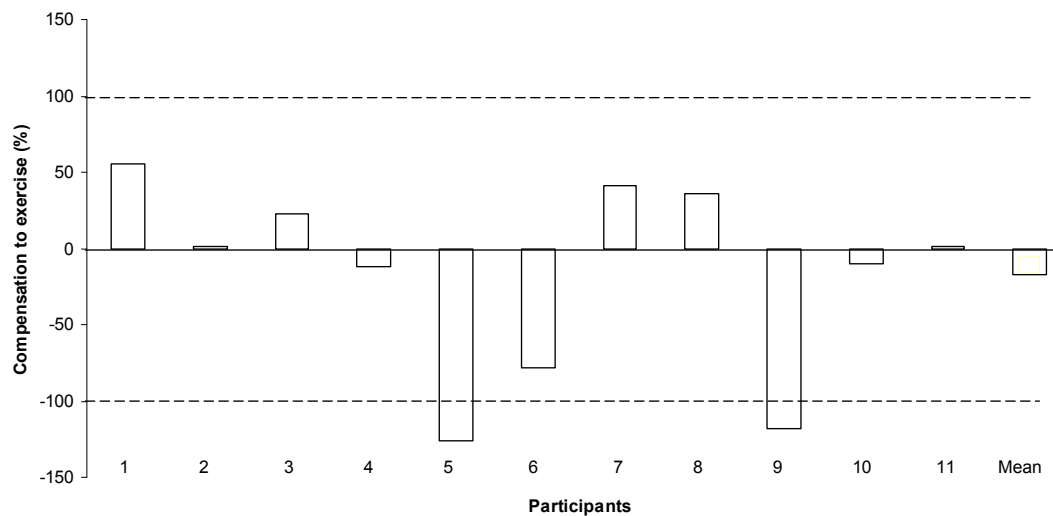


Figure 6.5 Compensation of exercise-induced energy expenditure during a buffet meal. Compensation =  $[(\text{energy intake during the buffet meal in each exercise trial} - \text{energy intake in control rest trial}) / \text{exercise cost of exercise above resting energy expenditure}] \times 100$ . A value of 0% represents energy intake after exercise being equivalent to energy intake after the rest trial; a value of 100% represents energy intake after exercise being 100% greater than after rest and this is equivalent to the energy expended in the exercise session above rest; a value of -100% represents energy intake after rest being 100% greater than after exercise and is equivalent to the energy expended in the exercise session above rest (see general methods for more details).

Figure 6.5 presents compensation to exercise. Mean compensation was -17%. From the Figure 6.5, it can be seen that over 50% of participants ate less following exercise and energy intake was less than energy expended in the exercise session. Energy intake following exercise was similar to following rest in four participants. The compensation of the exercise-induced energy expenditure was not related to circulating concentrations of hormones such as acylated ghrelin, leptin, total PYY and PP.



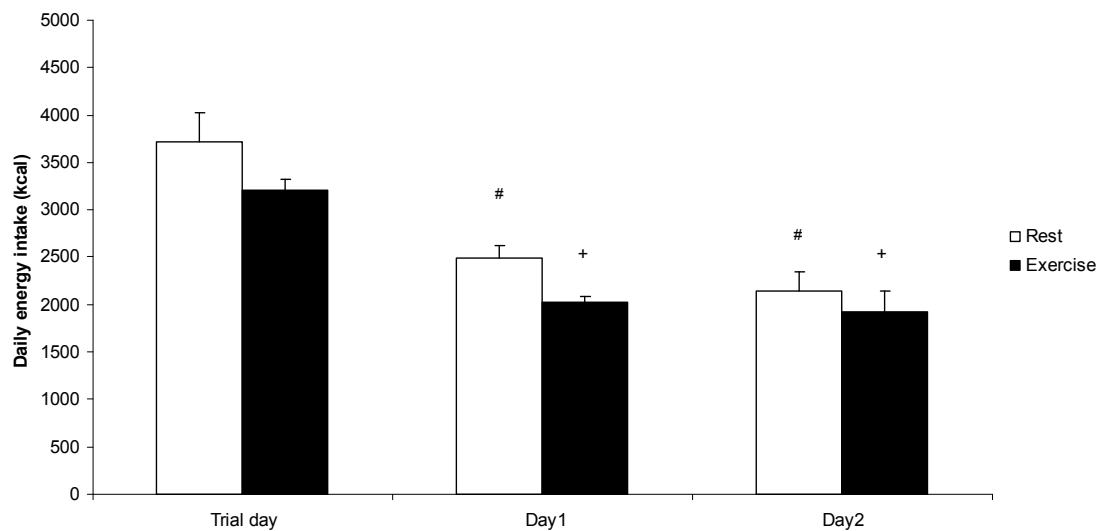


Figure 6.6 Energy intake (EI) in rest and exercise trials. EI from a buffet meal and weighed food and fluid records on each trial day; Day 1, one day after each main trial; Day 2, two days after each main trial. Values are means $\pm$ SE (n=9). <sup>#</sup>, significantly different between trial day and day 1 or day 2 in rest trials. <sup>+</sup>, significantly different between trial day and day 1 or day 2 in exercise trials ( $P<0.05$ ).

Figure 6.6 illustrates energy intake three days estimated from weighed food and fluid records. There was no difference in energy intake during this period. There was an effect of time for daily energy intake ( $P<0.01$ ). Daily energy intake decreased significantly on day one and day two after rest and exercise trials ( $P<0.01$ ).

### 6.3.4 Subjective feelings of hunger and fullness

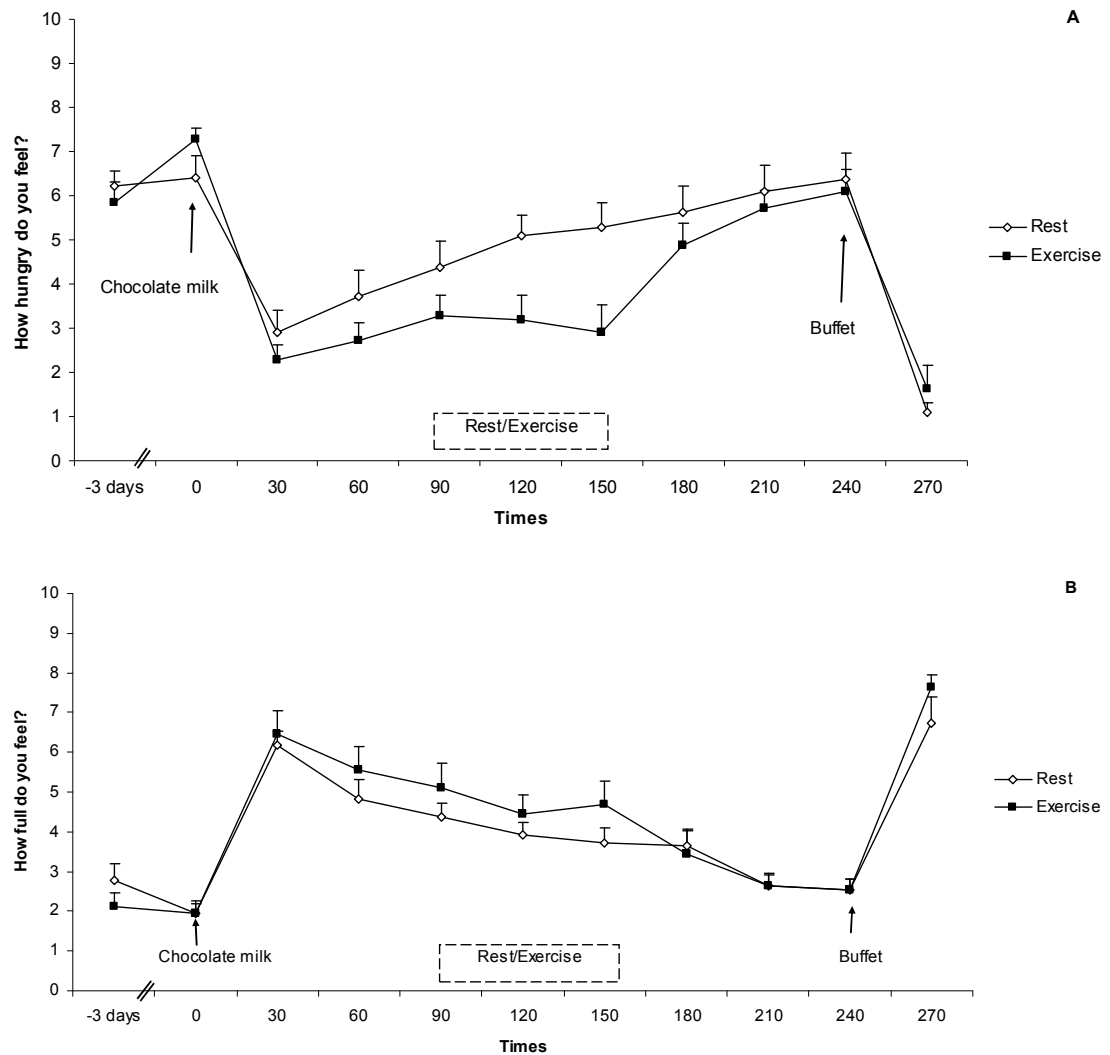


Figure 6.7 Subjective feelings of hunger and fullness using a 100 mm visual analogue scale (means $\pm$ SE) during exercise and rest trials (n=11). A, how hungry do you feel; B, how full do you feel. Data have been converted to a 0-10 scale for presentation.

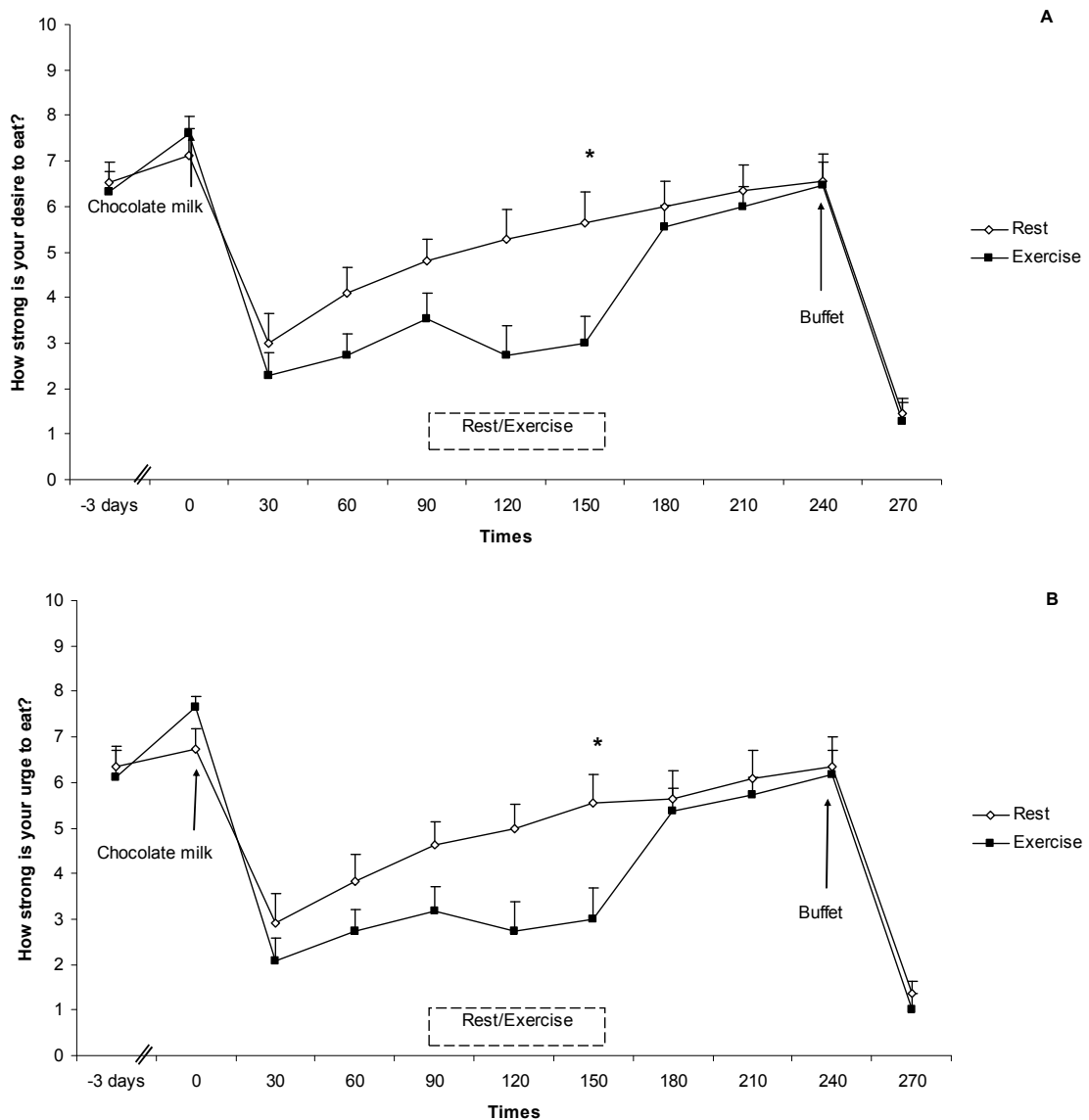


Figure 6.8 Subjective feelings of desire and urge to eat using a 100 mm visual analogue scale (means $\pm$ SE) during exercise and rest trials ( $n=11$ ). A, how strong is your desire to eat; B, how strong is your urge to eat. Data have been converted to a 0-10 scale for presentation. \*, significantly different between rest and exercise trials ( $P<0.05$ ).

Subjective feelings of hunger and fullness were assessed before and after food restriction as well as every 30 minutes on the main trial started from 0-270 minutes (Figures 6.7 and 6.8). It can be seen that subjective feelings of hunger and fullness did not differ significantly between trials at baseline, following 50% food restriction for three days and before rest or exercise (all  $P>0.13$ ). Two-ways ANOVA revealed a trial and time interaction for hunger ( $P=0.01$ ), desire ( $P=0.02$ ), and urge to eat ( $P=0.01$ ). There was no significant difference in hunger ( $P=0.07$ ) but there was a significant difference in desire ( $P=0.05$ ), and urge to eat ( $P=0.05$ ) immediately after exercise

between rest and exercise trials. There was a significant difference in desire ( $P=0.05$ ), and urge to eat ( $P=0.05$ ) immediately after exercise between rest and exercise trials and there was a trend for hunger ( $P=0.07$ ). There was a time effect for hunger, fullness, desire and urge to eat, as a result of consumption of chocolate milk and a buffet meal ( $P<0.01$ ). For clarity of presentation, these have not been depicted in the figures.

### 6.3.5 Physical activity energy expenditure

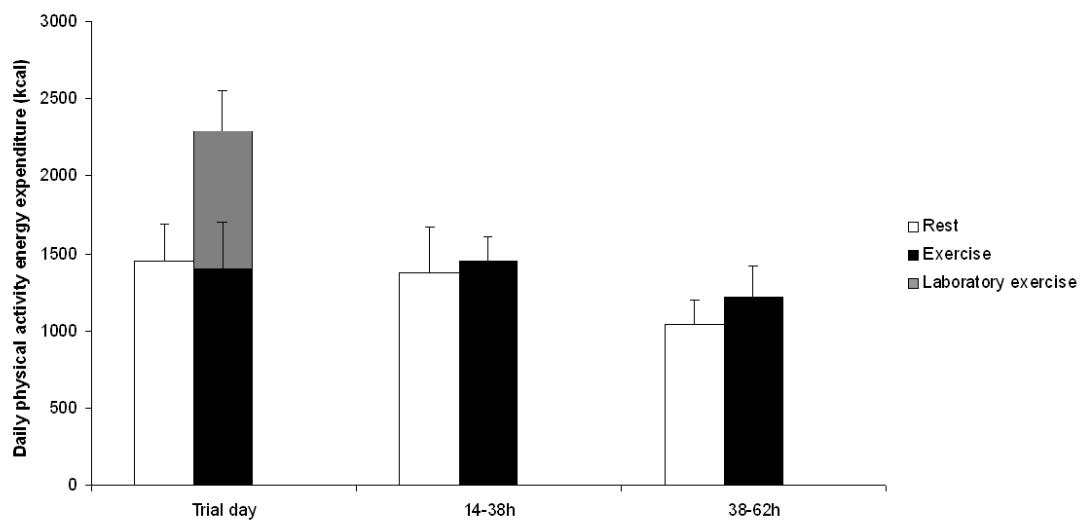


Figure 6.9 Physical activity energy expenditure (PAEE) in rest and exercise trials. Values are means  $\pm$  SE ( $n=9$ ). Trial day, rest or exercise; 14-38 h, between 14-38 hours; 38-62h, between 38-62 hours after main trials.

Figure 6.9 presents energy expended above rest (physical activity energy expenditure). There was no difference in physical activity energy expenditure between trials after subtraction of the energy spent during exercise in the laboratory.

## 6.4 Discussion

This study investigated the effect of acute high intensity exercise after eating on postprandial energy balance hormones in active males. The main hypothesis was that circulating postprandial hormones such as total PYY, PP and leptin concentrations might increase while acylated ghrelin would not change following high intensity exercise. Additionally, post-exercise energy intake following exercise would be lower than resting. Interestingly, circulating postprandial total PYY and PP concentrations were elevated for one hour following high intensity exercise. However, there was no impact of exercise on circulating postprandial acylated ghrelin and leptin concentrations. Moreover, there was no difference in post-exercise energy intake during a buffet meal between trials although by this time there was no difference in circulating total PYY, PP, leptin and acylated ghrelin concentrations.

The main finding of this study is that, in the fed state, circulating postprandial total PYY concentrations were elevated immediately following high intensity exercise and remained elevated for one hour. Previous studies have shown that, in the fed state, circulating postprandial total PYY concentrations were slightly increased during and immediately after intermittent moderate intensity exercise (Martin et al. 2007; Ueda et al. 2009). The consumption of a test meal after high intensity exercise results in an increase in total PYY concentration (Broom et al. 2009). Another study demonstrated that postprandial PYY<sub>3-36</sub> concentrations tended to increase following exercise at 60% of maximum oxygen consumption (Cheng et al. 2009). It has been demonstrated that meal composition, especially fat, influences circulating total PYY concentrations (Lin et al. 2003). Despite the fat in the chocolate milk in this study being similar to a previous study (Martin et al. 2007), we showed a more pronounced impact on postprandial total PYY concentrations following high intensity exercise. Recent studies indicated that circulating postprandial total PYY concentrations are elevated immediately after high intensity exercise in a fasted state (Broom et al. 2009; Chapter 5). It appears that high intensity exercise after eating in this study induced greater elevations in circulating postprandial total PYY concentrations than intermittent moderate intensity exercise (Martin et al. 2007) and longer elevations than exercise in the fasted state (Broom et al. 2009 and Chapter 5). Ueda et al. (2009b) reported cycling for 30 minutes at high

intensity exercise leads to increase in PYY<sub>3-36</sub> concentrations for 30 minutes longer than moderate intensity exercise in healthy men. It is possible that type, duration and intensity of exercise influence PYY concentration. However, it should be noted that the changes in circulating postprandial total PYY concentration after exercise in this study may have been influenced by the previous three days food restriction. It appears that high intensity exercise contributed to the increase in circulating postprandial total PYY concentrations in active males.

In this study, circulating postprandial PP concentration was higher than resting immediately after exercise and it remained elevated for one hour. This data is consistent with the recent finding that postprandial PP concentrations increased following moderate intensity exercise and incremental exercise (Martin et al. 2007; Slwowski et al. 2001). It appears that the postprandial PP concentrations increase in response to both moderate and high intensity exercise.

There was no change in circulating postprandial serum leptin concentration after exercise in the present study. This is consistent with previous study showing no change in postprandial leptin concentration following moderate exercise (Cheng et al. 2009). However, one study reported that postprandial leptin concentration was elevated following incremental exercise (Sliwowski et al. 2001). Some studies demonstrate that serum leptin concentrations were decreased following running and cycling for two hours in a fasted state (Duclus et al. 1999; Landt et al. 1997). It appears that there is no change in circulating postprandial leptin concentration after exercise in fed state.

Circulating postprandial acylated ghrelin concentrations after exercise did not change in this study. These data are consistent with a recent finding (Broom et al. 2009). Additionally, previous studies demonstrated there was no change postprandial total ghrelin concentrations after exercise (Martin et al. 2007; Ueda et al. 2009a). However, some studies demonstrated that acylated ghrelin concentration was suppressed following high intensity exercise in a fasting state compared with rest (Broom et al. 2009; Broom et al. 2007; Chapter 5). It appears that circulating acylated ghrelin concentrations may respond to acute high intensity exercise in the fasting state rather

than in fed state. To sum up, there was no impact of high intensity exercise after eating on postprandial acylated ghrelin in active men.

Despite the trend for absolute energy intake in a buffet meal after exercise to be lower than resting, there was no statistical difference between trials. This is consistent with a previous study in this thesis indicating that there was no difference in absolute energy intake in a buffet meal following exercise (Chapters 4 and 5). A recent study (Ueda et al. 2009a) showed that energy intake during a buffet meal was decreased following exercise in a fed state compared with rest whereas another study reported energy intake increased following exercise (Martin et al. 2007). Additionally, energy intake following exercise for two days was not different between trials in the present study. This is consistent with previous studies (Jamurtas et al. 2004, Chapters 4 and 5). To sum up, there was no change in post-exercise energy intake in this study.

In the present study, desire and urge to eat following exercise was lower than following rest. Previous studies showed no change in desire or urge to eat between trials in a fed state (Ueda et al. 2009a) and a fasted state (Chapters 4 and 5). It appears that exercise in fed the state induced suppression of the desire to eat.

The lack of change in energy intake might be explained by the fact that there was no difference in circulating postprandial total PYY, PP, leptin and acylated ghrelin concentrations at the time of the buffet meal. It is possible that if a buffet meal was provided immediately following exercise when total PYY and PP concentrations were greater than resting, energy intake following exercise might have been affected. Ueda et al. (2009b) reported post-exercise energy intake during a buffet meal after high intensity exercise was not different from moderate intensity exercise in the fed state even though there was a difference in PYY<sub>3-36</sub> concentration before a buffet meal. However, the findings in the present study suggest that the effect of exercise on total PYY and PP concentrations did not last very long.

Physical activity energy expenditure (PAEE) was not different between trials in active males in this study. This is supported by a previous study in this thesis demonstrating that there was no impact of high intensity exercise after an overnight fast in active men

(Chapter 5). In contrast, previous research has shown that PAEE can be increased for a few days following moderate intensity exercise in sedentary males (Chapters 3 and 4). One study reported that PAEE was increased after eight months of exercise training in sedentary males (Hollowell et al. 2009). Accordingly, it is clear that there was no effect of a single bout of exercise on PAEE following acute exercise in active young males in both fasted and fed states.

In this study, fasting total PYY, acylated ghrelin and leptin concentrations were decreased following three days of food restriction. This is consistent with previous studies showing that leptin concentrations decreased after a few days of food reduction (Eric et al. 2004; Weigle et al. 1997; Mars et al. 2005; Bloom et al. 2006) and complete fasting (Chan et al. 2004). Acylated ghrelin concentrations decreased after food restriction in this study. A previous study showed total ghrelin concentration decreased after food restriction and exercise (Maestu et al. 2008). Some studies reported total ghrelin concentrations did not change after food restriction (Eric et al. 2004; Bloom et al. 2006). It should be noted that participants in this study were active and they also maintained their activity for two days during food restriction. Therefore, the impact of food restriction on total PYY, acylated ghrelin and leptin concentrations might be affected by activity combined with food restriction in active men. In the present study, plasma total PYY concentrations decreased after food restriction. This data is consistent with a previous study where PYY concentrations decreased after fasting (Chan et al. 2004). Accordingly, it appears that three days of 50% food restriction leads to a decrease in fasting total PYY, leptin and acylated ghrelin concentrations in active men.

There are some limitations in the present study. Firstly, all participants were active, and we might not see similar effects in sedentary or obese participants. Active participants were selected because we showed the impact of high intensity exercise on acylated ghrelin and total PYY concentrations in previous study (Chapter 5). Secondly, participants ate only chocolate milk before rest or exercise. Little is known about the effect of different types of meal. Finally, participants ate over 50% of their daily energy intake during a buffet meal. This might be one reason why we did not see the impact of exercise on subsequent energy intake.



In summary, circulating postprandial total PYY and PP concentrations were elevated following high intensity exercise. However, there was no impact of high intensity exercise on absolute energy intake, appetite, physical activity energy expenditure, and postprandial acylated ghrelin and leptin concentrations. This finding suggested that high intensity exercise after eating leads to a short-term increase in satiety hormones for one hour but that this has no impact on post-exercise energy intake using the method employed in this study.

## CHAPTER 7

### GENERAL DISCUSSION

#### 7.1 Introduction

Exercise increases energy expenditure directly and theoretically may also influence energy intake and physical activity energy expenditure indirectly (i.e., through changing the energy balance hormones that influence energy intake and physical activity energy expenditure). This thesis aimed to investigate the impact of a single bout of exercise on post-exercise energy intake, physical activity energy expenditure (PAEE) and energy balance hormones in sedentary and active men. In the first study, the effect of acute exercise and the short term effect of exercise intensity (moderate and high) on post-exercise energy intake, appetite, PAEE and energy balance hormones was investigated in sedentary males (Chapter 3). There was no impact of exercise on energy balance hormones and post-exercise energy intake (Chapter 3). Daily PAEE tended to increase between 38 and 62 hours after moderate exercise intensity in sedentary men although this was variable and data was only available for a sub-sample. This study led to the hypothesis that moderate intensity exercise at 40%  $\dot{V}O_2\text{max}$  may alter energy balance hormones such as leptin and adiponectin concentrations after exercise (i.e., 38-62 h). Consequently, the impact of a single bout of exercise at 40%  $\dot{V}O_2\text{max}$  on post-exercise physical activity energy expenditure and energy balance hormones after exercise was investigated in sedentary men (Chapter 4). There was no change in circulating energy balance hormone concentrations and post-exercise energy intake in sedentary men (Chapter 4). It was speculated that differences in the habitual physical activity level of participants may be important in governing the response to acute exercise and that more active individuals may show better regulation than less active individuals. Therefore, the impact of moderate and high-intensity exercise on energy intake and physical activity energy expenditure in active men was investigated (Chapter 5). It was found that plasma acylated ghrelin concentration decreased whereas total PYY concentration tended to increase after high intensity exercise in active men. Additionally, the response to a single bout of exercise might be affected by diet restriction and recent energy intake before exercise; and exercise for weight loss often takes place in the

context of some form of caloric restriction. As a result, the final study (Chapter 6) aimed to investigate the impact of exercise on postprandial circulating energy balance hormones concentration and post-exercise energy intake and physical activity energy expenditure in active men. It was found that total PYY and PP concentrations increased after high intensity exercise in active men.

## 7.2 The main findings

- Daily physical activity energy expenditure (PAEE) tended to increase between 38 and 62 hours after moderate intensity exercise at 40%  $\dot{V}O_{2\max}$  in sedentary men (Chapter 3).
- There was no impact of moderate and high intensity exercise on post-exercise absolute energy intake and physical activity energy expenditure following either moderate or high intensity exercise in active men (Chapters 5 and 6).
- A single bout of walking at 40%  $\dot{V}O_{2\max}$  induced positive changes in subsequent free-living physical activity energy expenditure over the two days following exercise in sedentary males. This was mostly accounted for by increased low-to-moderate non-exercise behaviour such as standing and walking in the 2.4 to 4.78 METs range (Chapter 4).
- An increase in PAEE following exercise cannot be explained by a systemic change in circulating energy balance hormones leptin, adiponectin and acylated ghrelin (Chapter 4).
- There were no significant differences in plasma acylated ghrelin and leptin concentrations and perceived feelings of hunger and fullness before a buffet meal, following rest or exercise in sedentary and active men (Chapters 4, 5 and 6).
- Circulating plasma acylated ghrelin concentration decreased while total PYY concentration tended to increase following high-intensity exercise in a fasted state in active males (Chapter 5).
- Postprandial plasma total PYY and PP concentrations were elevated for one hour following high intensity exercise in a fed state while there was no change in postprandial acylated ghrelin concentration in active males (Chapter 6).

- Fasting total PYY, acylated ghrelin and leptin concentrations were decreased but there was no change in PP concentrations after three days 50% food restriction (Chapter 6).

This research aimed to investigate the effect of a single bout of exercise (fasted and postprandial) on energy intake, physical activity energy expenditure and energy balance hormones in sedentary and active men. This thesis showed no impact of acute exercise of either moderate or high intensity on post-exercise energy intake during a buffet meal in sedentary and active men (Chapters 4, 5, and 6). The lack of effect on post-exercise energy intake agreed with the lack of effect on appetite (subjective feelings of hunger and fullness) and hormones which influence energy intake such as acylated ghrelin, leptin, total PYY and PP concentrations (Chapters 4, 5 and 6). Although this thesis shows that total PYY (Chapters 5 and 6) and PP (Chapter 6) increased whereas acylated ghrelin concentration (Chapter 5) decreased due to high intensity exercise, this effect was short-lived and had already disappeared by the time a buffet meal was provided one hour later. It is possible that if a buffet meal had been provided when there was a difference in the concentration of energy balance hormones, then energy intake following exercise might have been lower than resting.

Data from several chapters of this thesis was pooled in order to examine relationships between parameters (Table 7.1). There is an inverse correlation between post-exercise energy intake during a buffet meal and PP concentration before a buffet meal was provided (Chapter 6); however, there was no relationship between post-exercise energy intake and acylated ghrelin (Chapters 4, 5 and 6), leptin (Chapters 4, 5 and 6) and PYY (Chapters 5 and 6) concentrations (Table 7.1). Circulating PP does not cross the blood brain barrier and hence influences energy intake via a vagal pathway. If this relationship holds true, then this would suggest that manipulating PP (e.g. through exercise) would affect energy intake but only at the time when circulating concentrations of the hormones were affected.

Additionally, there was no relationship between post-exercise energy intake during a buffet meal and subjective feelings of hunger and fullness before a buffet meal (Chapters 4, 5 and 6). It is possible that the concentration of energy balance hormones

and subjective feelings of hunger and fullness poorly correlate with the energy intake during a buffet meal. Importantly, there was individually variability in energy intake between participants in response to exercise and some participants ate more than half of their daily total energy intake in this one meal (Chapters 4, 5 and 6). This might explain why most of the studies in this thesis fail to detect differences in post-exercise energy intake. It is possible that other factors influenced energy intake in this context rather than energy balance hormones and subjective feelings of hunger and fullness. Such factors might include the free access to food and the hedonistic aspect of food consumption when presented with a range of food choices.

Parameters	Leptin (n=43)	Acylated ghrelin (n=43)	Total PYY (n=21)	PP (n=11)	Energy intake
Rest					
VAS-1	0.24	-0.20	-0.07	0.18	-0.10
VAS-1	-0.12	-0.23	0.25	-0.34	0.16
VAS-1	0.17	0.26	-0.20	0.19	-0.67
VAS-1	0.19	0.23	-0.12	0.09	<0.01
EI (n=31)	0.04	-0.02	0.09	-0.56	-
Exercise					
VAS-1	0.3	0.08	-0.19	0.01	-0.12
VAS-1	-0.17	-0.15	0.36	-0.18	<0.01
VAS-1	0.19	0.17	0.07	-0.02	-0.12
VAS-2	0.13	0.18	-0.01	-0.09	-0.09
EI (n=31)	-0.07	-0.08	-0.08	-.67*	-

Table 7.1 Relationship between subjective feelings of hunger and fullness and energy intake during a buffet meal and energy balance hormones before a buffet meal. Data from Chapters 4, 5 and 6. EI, energy intake ; VAS-1, how hungry do you feel; VAS-2, how full do you feel. VAS-3, how strong is your desire to eat; VAS-4 , how strong is your urge to eat. \*, significant relationship ( $P<0.05$ ).

Interestingly, post-exercise energy intake during a buffet meal after high intensity exercise in the fed state (Chapter 6) was considerably less than after exercise in the fasted state (Chapter 5) in active men (Figure 7.1). Furthermore, it is interesting that there was no difference in post exercise energy intake between the two studies during a

buffet meal after addition of the energy intake from the chocolate milk before exercise (Chapters 5 and 6). Future work is required to explore the impact of prior feeding on energy balance hormones.

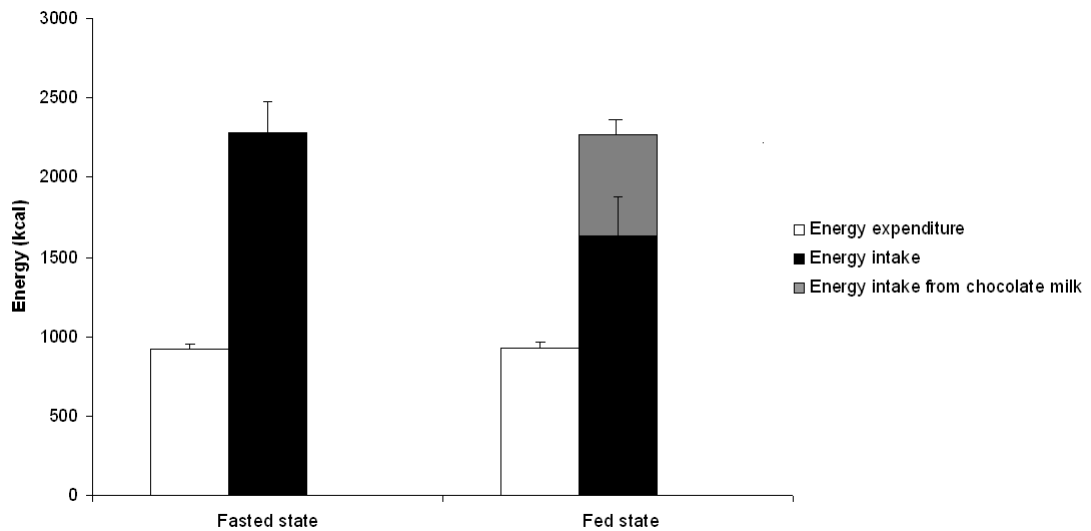


Figure 7.1 Energy expenditure in exercise and energy intake during a buffet meal in fasted and fed states. Data from Chapters 5 and 6.

There was no impact of either moderate or high intensity exercise on daily PAEE in active men (Chapter 5). Additionally, there was no impact of acute exercise on daily PAEE in a fed state after a few days of food restriction in active men. Interestingly, daily PAEE increased following moderate intensity exercise in sedentary males (Chapters 3 and 4). Little is known about how sedentary individuals respond to exercise in the fed state after food restriction. Therefore, further research might need to investigate the effect of exercise in a fed state after a few days of food restriction.

High intensity exercise after eating results in a transient increase in circulating satiety hormones such as postprandial total PYY and PP but there was no change in postprandial acylated ghrelin concentrations in active men (Chapter 6). In contrast, high intensity exercise after an overnight fast leads to the suppression of the circulating hunger hormone acylated ghrelin immediately after exercise in active men (Chapter 5) but not in sedentary men (Chapter 3). It is possible that the impact of exercise on energy balance hormones is influenced by pre-exercise fed or fasted state as well as the

physical activity level of participants. This finding would suggest that participants felt full after high intensity exercise in the fed state as well as feeling less hungry after high intensity exercise in fasted state. However, the data from subjective feelings of hunger and fullness did not show this pattern. It appears that the link between hormones and subjective feelings of hunger and fullness is unclear.

It is possible that altered circulating acylated ghrelin concentration might be influenced by either energy expenditure or physical activity level. Active men might have a greater response to high intensity exercise than more sedentary men. However, it is still not clear how much energy must be expended during exercise to suppress acylated ghrelin concentrations. It should be noted that if participants have to do high intensity as well as long duration exercise in order to suppress acylated ghrelin concentration then this might be difficult for sedentary or obese participants. Although ghrelin infusion leads to an increase in energy intake in humans (Wren et al. 2001), there is no relationship between plasma ghrelin concentration before a buffet meal and energy intake during a buffet meal (Table 7.1). Because we did not provide food when plasma acylated ghrelin was suppressed, we cannot conclude that suppression of acylated ghrelin was not related to post-exercise energy intake.

Table 7.2 presents relationships between energy balance hormones and participant characteristics and also subjective feelings of hunger and fullness at the baseline in this thesis. For instance, total percent body fat was highly correlated with fasting serum leptin concentration ( $r=0.77$ ,  $P<0.01$ ) and trunk fat ( $0.76$ ,  $P<0.01$ ). There is a relationship between energy balance hormones, for example, acylated ghrelin concentration is highly correlated with PP concentration ( $r=0.81$ ,  $P<0.01$ ). Subjective feelings of fullness tended to show an inverse relationship with serum leptin concentration ( $P=0.07$ ). Urge to eat tended to show a moderate correlation with plasma acylated ghrelin concentration ( $P=0.07$ ). Previous studies have shown a difference in ghrelin concentration between active and inactive participants (Jurimae et al. 2009; Jurimae et al. 2007a). However, in this thesis there is no relationship between fasting acylated ghrelin concentration and physical activity level as well as maximum oxygen consumption (Table 7.2).

Parameters	Leptin (n=43)	Acylated ghrelin (n=43)	Total PYY (n=21)	PP (n=11)
BW	0.32	0.17	-0.03	-0.43
BMI	0.38	-0.14	-0.12	-0.31
% Body fat	0.77*	-0.05	0.32	-0.28
Trunk fat	0.76*	-0.05	0.26	-0.16
Lean	0.12	-0.16	-0.21	-0.45
BMC	-0.02	-0.26	-0.34	-0.55
$\dot{V}O_2\text{max}$	-0.23	-0.2	-0.4	-0.03
PAL (N=31)	0.07	-0.13	-0.22	-0.28
Acylated ghrelin	-0.04	-	<0.01	0.81*
Total PYY	0.37 (n=21)	<0.01 (n=21)	-	-0.03
PP	-0.16 (n=11)	0.81 (n=11)	-0.03(n=11)	-
Leptin	-	-0.03	0.37	-0.16
Glucose	0.13	-0.19	0.12	-0.67
VAS-1	0.04	0.06	0.36	0.19
VAS-2	0.28	-0.18	-0.21	-0.35
VAS-3	0.01	-0.24	0.03	0.44
VAS-4	-0.13	0.28	0.07	0.23

Table 7.2 Relationship between energy balance hormones at baseline and other parameters. Data from Chapters 3, 4, 5 and 6. BW, body weight; BMI, body mass index; BMC, bone mineral content;  $\dot{V}O_2\text{max}$ , maximum oxygen consumption; PAL, physical activity level for seven days. VAS-1, how hungry do you feel; VAS-2, how full do you feel. VAS-3, how strong is your desire to eat; VAS-4, how strong is your urge to eat. \*, significant relationship ( $P<0.05$ ).

### 7.3 Limitations

Research into energy balance and human behaviour is very complex. It appears that previous findings from animal experiments might not readily translate to the human studies in this thesis. For example, spontaneous physical activity is increased due to leptin administration in mice (Hwa et al. 1997) whereas the increase in PAEE in sedentary men after moderate exercise was not associated with leptin concentrations (Chapter 4). Orexin A is released from the hypothalamus and injection into



paraventricular nucleus in rats causes an increase in spontaneous physical activity (Kiwaki et al. 2003). However, we cannot easily measure hormones in the brain which might influence energy balance. Another study showed that a change in hormones in mice and rats relates to the change in energy intake (Tschop et al. 2000). One of the major challenges of using human participants relates to the accurate assessment of energy intake (both free-living and during a buffet meal). Participants in this thesis were limited to young men and even the sedentary participants were quite active. It is possible that we might not see similar effects in different groups such as obese participants.

#### **7.4 Future research**

This thesis shows the impact of high intensity exercise on some energy hormones in active men. However, little is known about the impact of the same intensity and duration in different groups. It would be interesting to examine whether acute exercise exerted the same effect when active men become inactive (or vice versa). Post-exercise energy intake after high intensity exercise in a fasted state appears to be similar to post-exercise energy intake in a fasted state after the addition of the energy intake from consuming chocolate milk before exercise. Future research is needed to investigate the effect of consuming different type of energy before exercise. We also know very little about the mechanisms involved in energy balance. Clearly, many of the mechanisms will require a greater understanding of changes induced within the brain by acute exercise and such studies are difficult to perform in humans.

#### **7.5 Conclusion**

The results in this thesis suggest that acute high intensity exercise contributes to the suppression of circulating hunger hormones (acylated ghrelin) as well as elevated satiety hormones (total PYY and PP) in active men, but there was no impact on behaviour in terms of either post-exercise energy intake and physical activity energy expenditure. An acute bout of exercise leads to a transient change in behaviour in sedentary participants (an increase in PAEE). This finding might support the use of exercise for the treatment or prevention of obesity. However, little is known about the

effect of exercise training and repeated bouts of exercise. This thesis is only one step towards improving understanding of the impact of exercise on energy balance and clearly further research is required.

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## Health questionnaire

Name.....Date .....  
Address.....  
.....  
Tel ..... E-mail address.....

*It is important that volunteers participating in research studied are currently in good health and have no significant medical problems in the past. This is to ensure that their own continuing well-being and to avoid the possibility of individual health issue confounding study outcome.*

**Please complete this question to confirm your fitness to participate and if you have any questions, please ask us.**

1. What is your date of birth?
2. Are you taking prescribed medication?  
☐ Yes  
☐ No
3. Are you taking non-prescription medicine (e.g. aspirin)?  
☐ Yes  
☐ No
4. Has your doctor ever said that you should not exercise because you have a heart Arrhythmia?
5. Have you ever been diagnosed as having any form of chronic or episodic condition (e.g. Asthma, Anorexia, Digestive problem, Hypertension, Heart diseases and type2 diabetes)?

6. Do you have current illness or have you had an illness in the last 6 weeks (e.g. cold or flu)?
  
7. On average, how many units of alcohol do you consume in one week?
  
8. Do you or have you ever smoked? (Please provide detail)
  
9. How many times do you undergo structured vigorous exercise (e.g. that makes you sweat) for 30 minutes or more per week?
  
10. How long do you exercise on average each time?
  
11. How much has your body weight (kg) changed in the last 6 months?
  
12. Have you taken part in any study which has taking blood sample in last 2 months?

Signature .....Date .....

## **Questionnaire before main trials**

**Name**.....**Date** .....**Code**.....

**Please complete this question**

1. How long did you sleep last night?
  
  
  
  
  
2. At what time did you finish your dinner last night?
  
  
  
  
  
3. Did you consume any alcohol yesterday?
  
  
  
  
  
4. Did you consume any caffeine yesterday (e.g. tea or coffee)?
  
  
  
  
  
5. Have you performed any strenuous exercise in the last few days?
  
  
  
  
  
6. Did you have breakfast this morning?
  
  
  
  
  
7. Did you have a cold or infection in the last 7 days?

**Signature** .....**Date** .....

## Visual analogue scales

Name.....Date ..... Code.....

Time: ..... A.M. / P.M./-before/after food or snack or drink

**Please read carefully:**

**Instructions:** Please mark on the line

**Example:**

**Time: 12.30 A.M. / ~~P.M.~~-before/after food or snack or drink**

Not at all hungry	_____	As hungry as I have ever felt
-------------------	-------	-------------------------------



**5. How hungry do you feel?**

Not at all hungry	_____	As hungry as I have ever felt
-------------------	-------	-------------------------------



**6. How full do you feel?**

Not at all full	_____	As full as I have ever felt
-----------------	-------	-----------------------------



**7. How strong is your desire to eat?**

Very weak	_____	Very strong
-----------	-------	-------------



**8. How strong is your urge to eat?**

No urge to eat	_____	Strong, want to eat now
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## **Consent form**

### **The effect of acute exercise on energy hormones**

**Researchers:**

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Dr Keith Stokes                Tel: 01225 384190 Email: k.stokes@bath.ac.uk

**Please initial box**

**1. I confirm that I have read and understand the information sheet dated..... (Version.....) for the above study and have had the opportunity to ask question.**

☐

**2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason and without my legal rights being affected.**

☐

**3 I agree to take part in the above study.**

☐

_____ Name of subject	_____ Date	_____ Signature
_____ Name of person taking consent (If different from research)	_____ Date	_____ Signature
_____ Research	_____ Date	_____ Signature

**1 copy for participant; 1copy for researcher**

Name.....Date .....Code.....

**Buffet meal**

Food offered	Weight before(g)	Weight after (g )	Total Weight (g)
White bread			
Brown bread			
Butter			
Strawberry jam			
Orange marmalade			
Honey			
Mayonnaise			
Nutella			
Cheddar cheese			
Sliced ham			
Tuna			
Sliced lettuce			
Sliced tomato			
Sliced carrots			
Apple			
Orange			
Banana			
Orange juice			
Coca-cola			
Water			
Coffee			
Tea			
Milk			
Sugar			
Chocolate ice-cream			
Vanilla ice-cream			
Kit-Kat			
Plain potato crisps			
Chocolate cookies			

## **BORG SCALE OF PERCEIVED EXERTION**

<b>6</b>	
<b>7</b>	<b>Very, Very Light</b>
<b>8</b>	
<b>9</b>	<b>Very Light</b>
<b>10</b>	
<b>11</b>	<b>Fairly Light</b>
<b>12</b>	
<b>13</b>	<b>Somewhat Hard</b>
<b>14</b>	
<b>15</b>	<b>Hard</b>
<b>16</b>	
<b>17</b>	<b>Very Hard</b>
<b>18</b>	
<b>19</b>	<b>Very, Very Hard</b>
<b>20</b>	

## Some food recording guidelines

### ***Instructions***

You must weigh and record your food and fluid intake for 2 days before the first main trial (rest or exercise), on the day of this trial and 2 days after main trial. During your second trial (rest or exercise), you must repeat your diet and consume the same foods and fluids as during your first trial.

### **Remember:**

1. Do not consume any alcohol during the two days before each trial, and during the day after the trial.
2. Do not consume any food supplements during this time (e.g. multivitamins).
3. When weighing food, do not forget to subtract any food that you have left over after your meal. This includes things such as apple cores, banana skins etc...
4. The dietary analysis is only ever as accurate as the accuracy of the food record itself. It is therefore highly important that care is taken to record the amount and type of food eaten, any method of cooking used, characteristics (e.g. whether fresh, tinned, dried etc) and timing and frequency of consumption, as these details will all have an effect on the overall output data and quality of dietary assessment.

**N.B All packet foods, tins etc. give weights on the side of the packaging**

<b>Fluid</b>	<b>Glass(1/3 pint) = 200g</b>
<b>Bread</b>	<b>Slice = 35 g</b>
<b>Rolls</b>	<b>50 g each</b>
<b>Butter</b>	<b>10 g/ pat</b>
<b>Honey</b>	<b>20 g/ slice</b>
<b>Eggs</b>	<b>60 g</b>
<b>Sausage</b>	<b>60 g</b>
<b>Meat/fish medium portion</b>	<b>120 g</b>
<b>Cheese- 2matchbox pieces</b>	<b>60 g</b>
<b>Vegetables</b>	<b>2 tbsp = 90 g</b>
<b>Green salad /lettuce</b>	<b>3 tbsp = 90 g</b>
<b>Potatoes</b>	<b>Medium = 200 g</b>
<b>Pasta</b>	<b>Large portion = 350g</b>
<b>Rice</b>	<b>Large portion = 200 g</b>



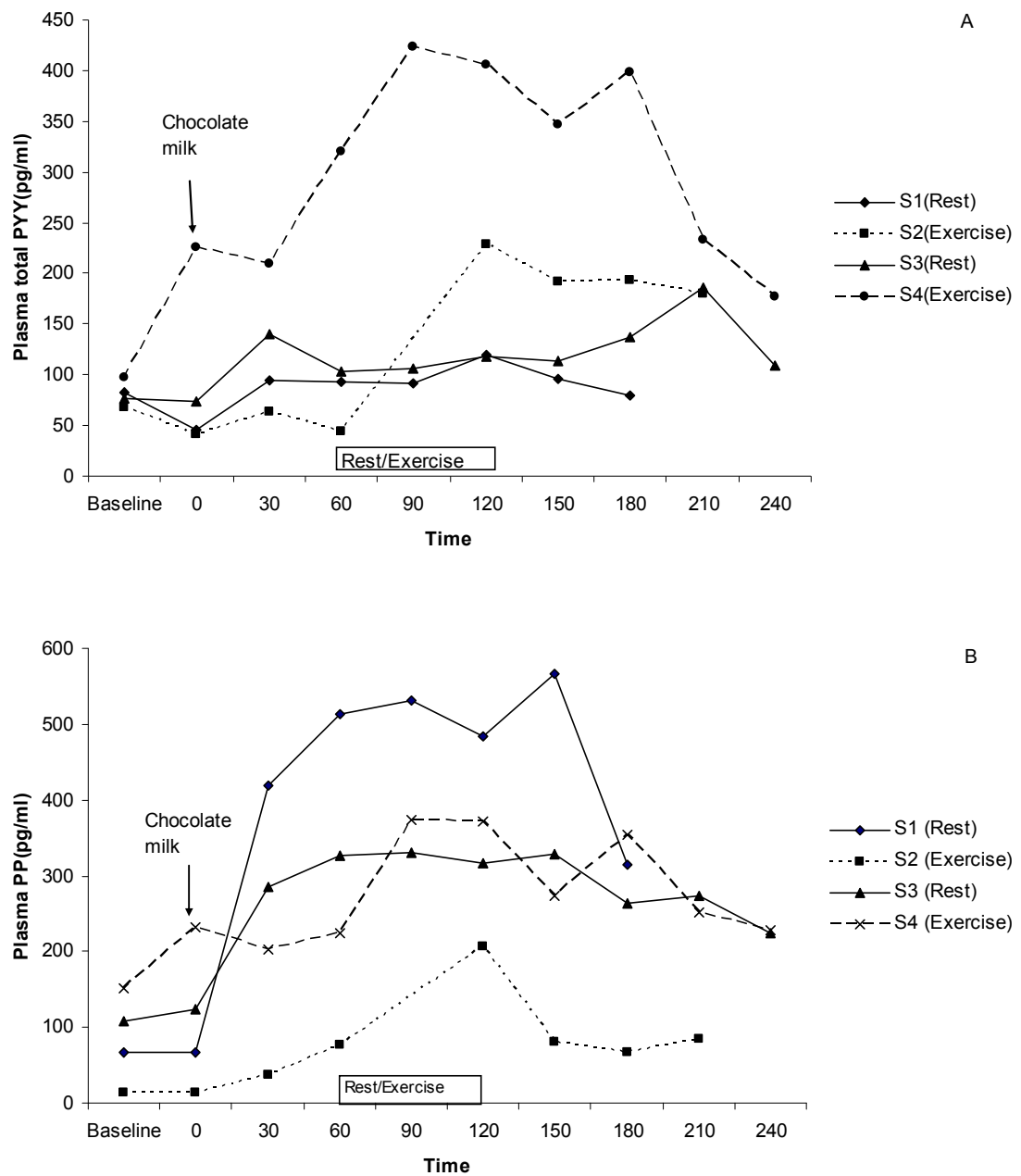


Figure (A) Plasma total PYY and (B) PP concentrations in pilot study 4 (Chapter 6).

Figures A and B compare total PYY and PP concentrations between rest and exercise in the pilot study in Chapter 6. There was a trend for total PYY concentrations to decrease after three days of food restriction; there was no change in PP concentrations.

Circulating postprandial total PYY and PP concentrations both during and after high intensity exercise tended to be higher than at rest.

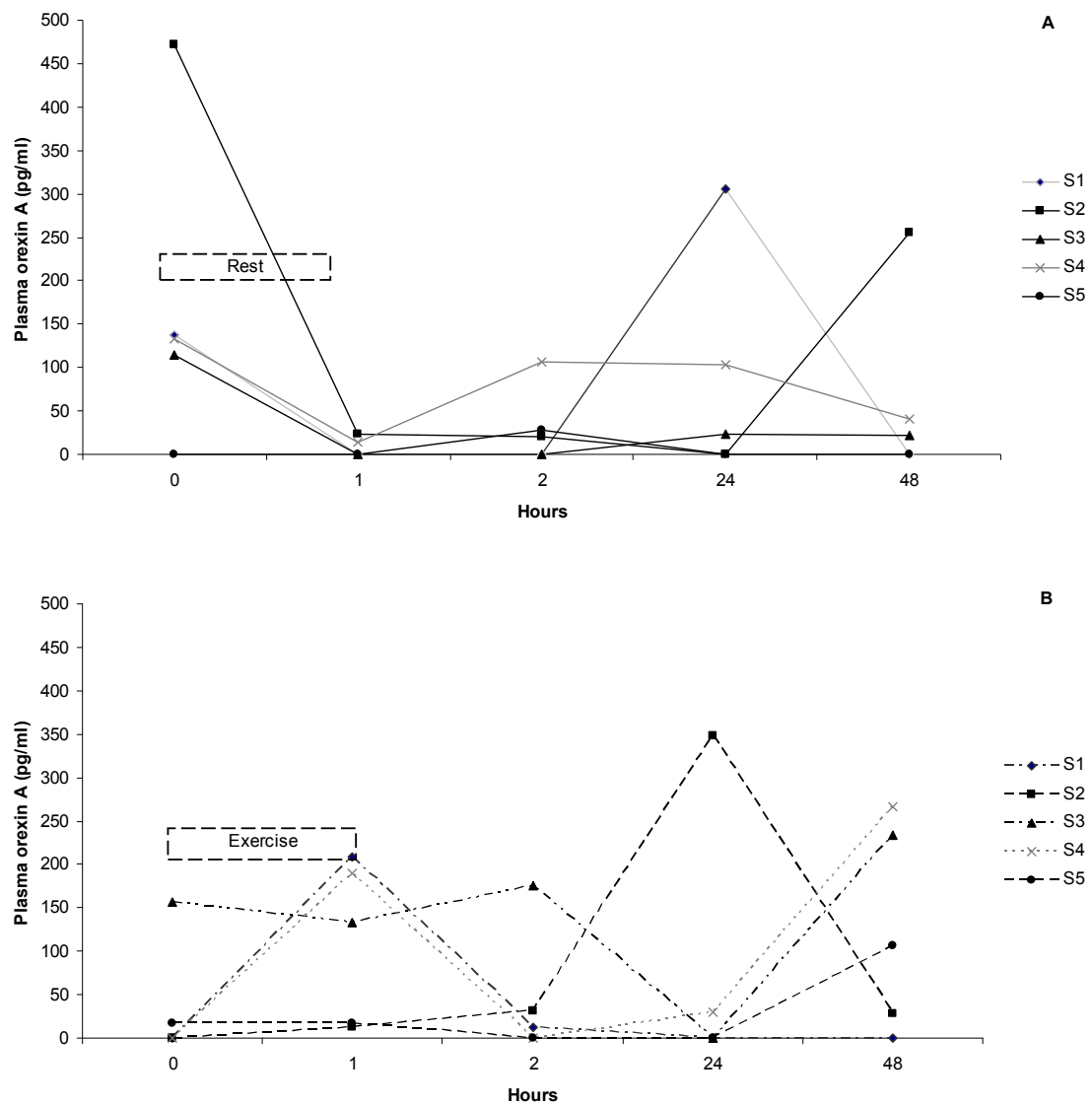


Figure Plasma orexin A concentrations (mean $\pm$ SE) during the rest (A) and exercise (B) trials (n=5).

There was no difference in plasma orexin A concentrations between rest and exercise trials. Orexin A was undetectable in most of samples.